ARCHIVES OF OPHTHALMOLOGY.

THE CLINICAL VALUE OF OPTIC CANAL ROENTGENOGRAMS.[1]

BY DR. HARRY A. GOALWIN, NEW YORK.[2]

(With eight illustrations on Text-Plates I. and II.)

IF we were to judge by the rarity with which optic canal Roentgenograms are called for by the ophthalmologist and neurologist we should be forced to the conclusion that the Roentgenography of the optic canal is difficult or impossible, or that the picture when obtained is of no clinical value. As a matter of fact there is, with the possible exception of the sella turcica, no deep structure of the skull which can be as easily and as clearly brought out and as unmistakably identified on the X-ray picture as the optic canals. Nor is there any structure in which the slight changes demonstrated Roentgenographically have as much bearing on the diagnosis or prognosis as they do when we are dealing with the optic canals.

The technique of the *precise* Roentgenography of the optic canals cannot be gone into here. It has been described by me in detail before the Roentgen Society of Vienna (April 8, 1924) (10). But as the principles of this technique have a direct bearing on the clinical value of the pictures obtained they will be briefly summarized:

I. A good, clear picture of the optic canals can only be ob-

[1] Read before the Section on Ophthalmology, New York Academy of Medicine, May 18, 1925.

[2] The author's researches on this subject are a part of his work as Du Bois Fellow of Columbia University for 1923–1924.

tained when we so place the head, X-ray tube and plate that the so-called central ray coincides with the axis of the optic canal and strikes the plate at right angles. It is only when these conditions are satisfied that we can draw conclusions from the picture as to the *shape* of the cross section of the canal or use the plate measurements of the optic canal to determine by calculation (or graphically) the actual dimensions of the structure in the skull. This means that we must know the *direction* of the axis of the optic canal in each case before we can make its Roentgenogram. Unfortunately (for the Roentgenologist) this direction is not the same in all individuals, as I have shown in my previous work (11). It varies with the age and to some extent with the race, and deviates considerably from the normal in various skull deformities.

II. Whatever the direction of the axis of the optic canal may be in a normal individual of a given race and age, its continuation *invariably* strikes the lower outer angle of the margin of the orbit. It is therefore there that we should expect to find it in a properly taken optic canal Roentgenogram. (Fig. 1.)

III. As the cross section of the optic canal is not circular, its size should be given by stating at least two of its diameters. The two principal diameters (referred to later as the "A" and "B" diameter) are *A*—directed downward and outward at an angle of 45 degrees, and *B*—directed at right angles to "A".

IV. An optic canal Roentgenogram is practically worthless unless it is accompanied by the following data: (1) the size of the focal spot of the X-ray tube used for taking the picture, (2) the distance of this focal spot from the plate and (3) the distance of the optic canal from the plate.[1] These data are necessary in order to determine the actual size of the optic canal from its Roentgenographic dimensions, by means of graphics or calculation. The description of a few Roentgenograms of optic canals of dried skulls will illustrate the principles of the precise Roentgenography of this region and help us to orient ourselves on the plates of the clinical cases given later.

[1] As the actual determination of this distance in the living is extremely difficult, the average distance for the age of the patient in question is taken from the curves which (11) I worked out from skull measurements.

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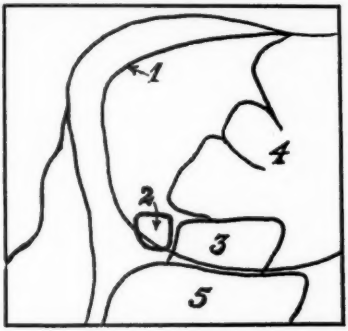
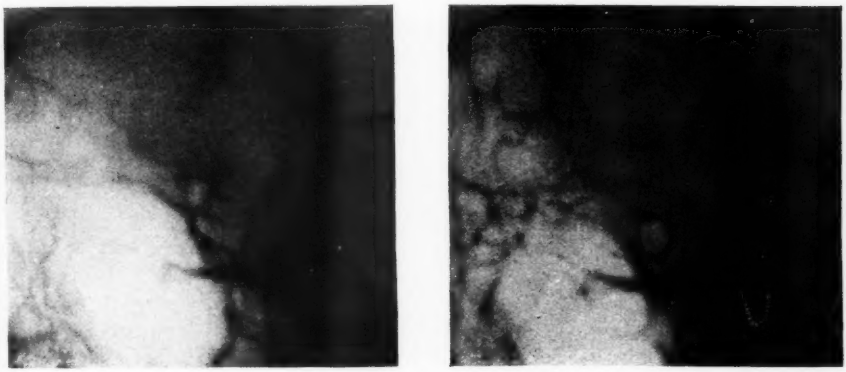


FIG. 1. Sketch showing relations of structures seen on optic canal Roentgenogram.



A B

FIG. 2. A. Apparent constriction of right optic canal, produced by faulty projection. B. Correct projection, showing same canal to be normal in shape and size. As this is from a case of post-meningitic optic atrophy, the first Roentgenogram would have led to a wrong prognosis. Patient has completely regained sight.

FIG. 3. Constriction of right optic canal by multiple osteomata of the orbit (Case 1).

FIG. 4. Marked thickening and increased density of walls of left optic canal by periostitis (Case 3).

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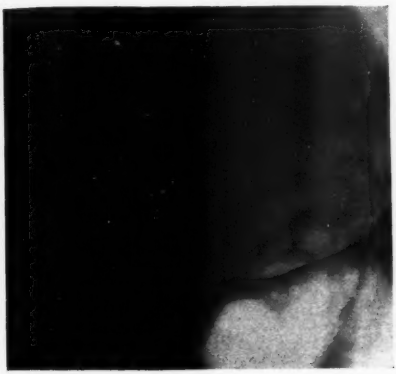
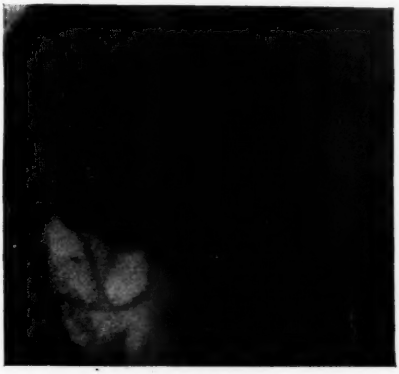
FIG. 5. Hyperostosis and exostosis of left optic canal following inflammation of pneumatized anterior clinoid process (Case 5).

FIG. 6. Destruction of roof of optic canal by tumor of middle fossa (Case 7).



RIGHT



LEFT

FIG. 7. Enlargement of left optic canal by optic nerve tumor. Right optic canal is shown for comparison (Case 9).

FIG. 8. Fracture of left optic canal, with displacement of the fragment toward the sphenoidal fissure (Case 10).

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The principal structures seen on these pictures are (see Fig. 1) (1) the orbital margin, (2) the optic canal, (3) below and mesial to the optic canal—a rhomboid structure which is an oblique projection of the sphenoidal sinus, (4) above and mesial to the optic canal—an oblique projection of the frontal sinus and (5) below the inferior orbital margin—an oblique projection of the maxillary sinus.

The focal spot of the tube used for the following four Roentgenograms was 3.00*mm* in diameter.

No. 1. Roentgenogram of the right orbit of a normal three-year-old child. The optic canal is projected at the lower outer angle of the orbital margin. It has the shape of the quadrant of a circle, the straight lines forming the roof and inner wall, the arc forming the floor and outer wall. Its *A* diameter is 5.5*mm* on the plate and 4.6*mm* on the skull. Its *B* diameter is 6.0*mm* on the plate and 4.5*mm* in the skull. The focus-plate distance is 52.75*cm*, the canal-plate distance is 5.4*cm*.

No. 2. Roentgenogram of the right orbit of a normal adult. The optic canal is projected at the lower outer angle of the orbital margin. Its shape approximates that of the quadrant of a circle, the straight lines forming the roof and inner wall, the arc forming the floor and outer wall. Its *A* diameter is 5.8*mm* on the plate and 4.50 in the skull. Its *B* diameter is of the same size. The focus-plate distance is 52.75*cm*, the canal-plate distance is 6.25*cm*.

No. 3. Roentgenogram of the right orbit of an adult scaphocephalus. The optic canal is projected in the lower outer quadrant of the orbit, at a point which is situated at a distance from the inferior margin equal to one third of the height of the orbital opening and a distance from the outer margin equal to one third of the width of the orbital opening. Its shape is somewhat triangular. There is a marked constriction of the *A* diameter which measures 3.3*mm* on the plate and 2.3*mm* in the skull. The *B* diameter measures 5.5*mm* on the plate and 4.3*mm* in the skull. Focus-plate distance 52.75*cm*. Canal-plate distance 4.6*cm*.

No. 4. Roentgenogram of the right orbit of an oxycephalus (Turmschädel). The optic canal is projected as in No. 3. In its shape it resembles a triangle with the angles rounded off.

Its *A* dimeter is 4.8*mm* on the plate and 3.5*mm* in the skull. Its *B* diameter is 5.5*mm* on the plate and 4.2*mm* in the skull. The focus-plate distance is 52.75*cm*; the canal-plate distance is 5.5*cm*.

It may not be amiss, before going into the details of the interpretation of optic canal Roentgenograms, to reiterate a few of the well-known fundamentals of skull Roentgenography in general.

The soft intracranial structures and their pathological changes, such as hemorrhages or collections of pus in the brain and meninges, cannot, except in very rare instances, be directly demonstrated on the X-ray picture.

Of the normal soft intracranial structures the only one that can often be directly demonstrated on the X-ray picture is the calcium containing pineal gland (Schüller) (2).

The only pathological change of the soft intracranial tissues that can be directly demonstrated on the X-ray picture is calcification, such as calcification of a sclerosed blood vessel, of an aneurismal *dilatation or of degenerating* tissue in a tumor or in an abscess.

We must therefore content ourselves with judging of the pathological changes of the soft intracranial content from the changes which they produce in the skull, which latter are directly recognizable on the X-ray picture.

These fundamentals apply, of course, to changes in the contents of the optic canal. Inflammation, hemorrhage or soft tumors in this region cannot be directly demonstrated on an optic canal Roentgenogram; bony tumors and calcifications can be directly demonstrated.

We must therefore be satisfied, in optic nerve disturbances, with judging of the underlying process from the changes which latter produces in the bony optic canal, when such changes are directly demonstrable on the X-ray picture.

A systematic and complete analysis of these optic canal changes consists of the following determinations:

1. Shape of the cross section of the optic canal.
2. Dimensions of cross section.
3. Length of canal.
4. Changes within the lumen.
5. Structure of the walls.

6. Changes in the adjacent structures.

It is difficult, indeed, almost impossible, to study any one of these elements without reference to one or more of the others; since a change in one usually involves a change in one or more of the others. Thus for instance, a constriction of an optic canal may be associated with a change in shape or a concentric thickening of the walls, or a chronic sphenoiditis, etc.

SHAPE OF THE CROSS SECTION.

This usually, in most of the normal adults as well as in most of juvenile skulls, closely approximates a quadrant of a circle, the limiting radii of which form the roof and the inner wall and the arc of which form the floor and outer wall. It often varies from this shape in the normal skull, being more or less elliptical, circular or even quadrangular, but never presenting a definite, regular geometrical figure.

Typical changes in the shape of the optic canal occur in certain skull deformities, in which it is usually associated with a reduction in size of one or both of the principal diameters of the cross section. The most important of these are the changes in the shape of the optic canal found in oxycephaly and scaphocephaly, since these conditions are usually associated with complete or partial optic atrophy. The optic canal Roentgenogram in most cases of oxycephaly shows a cross section which is distinctly triangular, the base of the triangle being directed downward and outward and being shorter than the altitude which is, of course, directed at right angles to it.

A similar change of shape of cross section is found in scaphocephaly, the ratio between the base and the altitude of the triangle being still smaller than in oxycephaly.

Other changes in shape are those associated with variations in the extent of pneumatization of the structures surrounding the optic canal (9), with chronic inflammation of the adjacent structures, with tumors of the orbit or tumors of the sella or anterior or middle fossa of the skull, with exostosis or with fractures.

The effect of excessive pneumatization of the structures adjacent to the canal have been described in detail by White.

According to this author an increase in the pneumatic space adjoining any part of the wall of the canal results in a flattening of the corresponding part of the wall. We have been unable to confirm this conclusion either in our inspection of skulls in the skeleton or by Roentgenography. The roof, which is most commonly flat, has no pneumatic space over it. The outer wall, however, bulges into the lumen in cases in which pneumatization of the anterior clinoid process is demonstrable on the X-ray picture (see Fig. 5). The triangular optic canals in the deformed skulls mentioned, with their nearly straight walls, show no constant, or even occasional associated excessive pneumatization of the adjacent structures.

The effect of chronic inflammations on the shape of the optic canal depends on the site of the inflammation. The effect of tumors on the shape of the optic canal depends on whether they are soft or bony and whether they are situated within the orbit, within the orbital wall within the anterior or middle fossa of the cranial cavity or within the sinuses.

Soft tumors cannot be directly demonstrated on the X-ray picture. They can only be recognized indirectly by the bone erosion or bulging they produce. The roof of the optic canal being its thinest part, soft tumors over it would be expected to produce bulging before actual erosion would begin. The anterior clinoid process being very rigid, tumors pressing upon it produce erosion rather than bulging into the lumen of the canal.

A soft tumor situated directly behind the optic canal will erode the posterior portion of the canal. As this portion is the narrowest one and has the cross section which gives the canal its typical shape on the Roentgenogram, the optic canal will appear on the Roentgenogram to be circular and increased in cross section.

Tumors of the sella often extend around the side of the optic canal to the bridge between the canal and the sphenoidal fissure. Here they may erode the bridge entirely. In a case of this kind which I saw in Vienna last spring the bridge was broken through, thus connecting the two spaces into one, which had the shape of a collar button, the head of which was the cross section of the optic canal, the stem—the defect in the bridge and the base—the inner part of the sphenoidal fissure.

Tumors situated within the sphenoidal sinuses may produce bulging of the inner wall of the canal into the lumen or break through this wall entirely.

Bony tumors are directly demonstrable on the X-ray picture (see Fig. 3). If situated directly along the axis of the optic canal they will obscure the optic canal on the Roentgenogram entirely or in part, depending on the extent to which the shadow of the tumor overlaps the shadow of the lumen of the optic canal. If situated in the orbital wall or in the optic canal wall they will encroach upon the lumen. They will similarly encroach upon the lumen when they are situated within the sphenoidal sinus. It must be kept in mind that a shadow of a bony tumor may be projected into the shadow of the lumen of the optic canal because it simply happens to lie along the path of the rays which produce the lumen shadow. Such interfering shadows may be at a considerable distance from the optic canal. Their exact location must be determined from the lateral and axial views and such other views as may be necessary to establish their location.

Exostoses produce the same changes in the shape of the cross section of the optic canal as bony tumors of the wall.

The change in shape of the optic canal due to fractures extending through its walls is due mainly to overproduction of bone in the healing process. Displacement of the fragments is rare (see, however, Fig. 8), as these fractures are merely fissures which extend into the canal from fractures of the orbital wall or from basal fractures of the skull.

SIZE OF THE CROSS SECTION.

The actual dimensions, *i.e.* the length of the two principal diameters, should be determined from the X-ray picture by calculation or graphically. It is important to note in this connection that *the optic canal reaches the adult size in both principal diameters at the age of three years*, the *B* diameter having reached its full adult size at the age of one year. It follows, therefore, that after the age of three years the size of the cross section of the optic canal has no relation to the age, hence a small optic canal is just as significant in a child as it is in an adult. The average normal *A* diameter in the adult

optic canal is 4.3*mm* and the average *B* diameter is 4.4*mm*. The corresponding maxima are 5.45 and 5.57*mm*. The corresponding minima are 3.25 and 3.5*mm*.

Optic canals below the average size are the rule in cases of optic nerve involvement which is secondary to sinus infection or other focal infection. In cases of unilateral involvement it is the side with the smaller canal that is usually involved. This was first pointed out by White and corresponds with our observations. When these small canals are above the minima stated and show no changes in their walls the Roentgenogram simply shows that in case of inflammation the optic nerve would be greatly compressed and more likely to atrophy than it would if the canal were larger. If in such a case optic atrophy is already present the small size of the canal indicates an unfavorable prognosis.

If the reduction in size is associated with increased thickness or density of the wall, or both, it is an indication that the optic nerve atrophy was caused by compression of the nerve. If the concentric thickening and increased density of the wall is limited to the optic canal it is an indication that we are dealing with a localized chronic periostitis of the canal.

Acute periostitis cannot be demonstrated on the X-ray picture.

Marked constriction of the canal without any changes in the structure of the wall is found in oxycephaly and scaphocephaly. Here, however, it is associated with the typical change in shape described above. The constriction here is more marked in the *A* diameter than in the *B* diameter, as has already been mentioned.

STRUCTURE OF THE WALLS OF THE OPTIC CANAL.

The bony tissue making up the walls of the optic canal is rather thin throughout. The roof forms the thinnest part especially at its posterior margin where it is reduced to knife-edge thinness. It gets gradually thicker toward the front. Very careful Roentgenography may be required to bring the sharp posterior margin of the canal roof out on the X-ray picture at all, for if we miss that, we miss the narrowest part of the optic canal, which part is the most important from the clincial point of view.

The outer wall consists of the bridge between the canal and the sphenoidal fissure. It is the thickest but the least dense part of the bone surrounding the optic canal, yet is only about 2.0*mm* thick. Its average length (antero-posteriorly) is 6.69*mm* (11).

The inner wall is made up of the external wall of the sphenoidal sinus, which sinus may also extend under the floor of the canal.

Thickening of the walls may be concentric, in which case it constricts the canal, or excentric, in which case there is no constriction. In either case the thickened part has its density increased.

Uniform increase of thickness and density of the entire circumference of the optic canal suggests periostitis (see Fig. 4) of the canal. They must be differentiated from the so-called partial hyperostosis of the sphenoid (3).

Localized areas of thickening are indicative of extension of bone inflammation from a neighboring structure, in which case they are diffuse, or of tumor (bony), in which they are circumscribed.

Decreased density of the wall of the optic canals, without any decreased thickness, may accompany a similar change in the body of the sphenoid. The outline of the sphenoid in these cases, especially of the sella, is made out with difficulty on the X-ray picture, but it is well defined and uninterrupted. It looks like a phantom. This condition suggests an infiltrating process of the sphenoid, usually tuberculosis (Schüller).

Decreased thickness of the wall is usually localized and is suggestive of erosion of the wall of the canal at the site in question by a soft tumor. Mechanically the same effect can be produced by the pressure of an aneurismal dilatation of a blood vessel.

Such localized erosions usually take place from without the optic canal, since any proliferation of tissue within the canal would exert a more or less uniformly distributed pressure in all directions and erode the entire inner surface of the wall of the canal, producing an enlarged, circular cross section of the canal (see Fig. 7). This latter condition can be differentiated Roentgenologically from that enlargement of the cross section of the optic canal which is produced by destruction of its nar-

rowest posterior portion, by the profile view, if necessary with the assistance of the axial or occipito-glabellar view. The details of this differentiation belong to the domain of technical Roentgenology and will be discussed elsewhere.

Endotheliomata of the optic nerve sheath will produce a general hyperostosis of the optic canal walls with localized erosions from within, the erosion being caused by an invasion of the optic canal walls along the course of the vessels. We therefore have in this property of the endothelioma a ready means of diagnosing it from the Roentgenogram.

Excessive pneumatization of the sphenoid bone often manifests itself in an extension of the sphenoid sinus into the floor of the optic canal, occasionally extending even into the outer wall. The entire canal may indeed be surrounded by the sphenoidal sinus so that the optic nerve appears to be passing through the sinus (Onodi), but there is always a small radial septum present in this annular air space, indicating that it is an extention from the sinus we are dealing with (Schaeffer). Incidentally, an ethmoid cell which often usurps the relation of the sphenoid sinus with the optic canal may pneumatize the walls of the latter in the same manner.

The clinical importance of knowing that there is such an excessive pneumatization has been pointed out by White, Schaeffer and others. The optic canal Roentgenogram will show this condition, and the axial view of the skull will show just which of the sinuses is at fault. Of still greater importance from the standpoint of prognosis in these cases is it to demonstrate Roentgenographically whether, after an acute or chronic involvement of these pericanalicular pneumatic spaces, the canal walls were left in a state of thickening or sclerosis or both, for such thickening and sclerosis will leave the canal deformed in its shape and constricted in its lumen. An atrophic optic nerve lying in such a canal has little chance for spontaneous recovery or favorable response to medication. Nothing short of decompression of the nerve can even give any hope of benefit.

The *length of the optic canal* can be determined from the profile Roentgenogram.

This view should give us information as to those fractures, which because of their parallel direction to the cross section of

the canal cannot be demonstrated on the posterior-anterior view. It is obvious that an inflamed optic nerve lying within a long canal is more likely to atrophy than one situated within a shorter canal of the same cross section. The likelihood to atrophy, in other words, is directly proportional to the length of the canal and inversely proportional to the size of the cross section.

Shadows within the lumen of the optic canal are usually extracanalicular, that is, they are projected from some point in front or behind the canal which happens to lie along the continuation of the optic canal axis. Intracanalicular shadows representing calcification may be found in cases of optic nerve atrophy (see Fig. 4) or in cystic tumors. In the former case there may be associated evidence of periostitis of the optic canal and in the latter case there may be associated evidence of optic nerve tumor (enlarged, circular cross section of optic canal).

The extracanalicular shadows within the lumen have already been discussed.

It is, of course, necessary to make a careful X-ray study of the entire skull whenever an investigation of the optic canals is undertaken. For just as the ophthalmoscopic picture of retinitis diabetica must be confirmed by glycosuria and hyperglycæmia, so must an optic canal deformity be confirmed by evidence of skull deformity or local pathology. Likewise is it necessary in cases of hyperostoses of the optic canal to establish whether it is associated with a general hyperostoses, such as is found in Paget's disease or acromegaly, etc., or with a partial hyperostoses limited to the sphenoid bone, or, finally, whether we are dealing with a purely local inflammatory reaction or a tumor.

The foregoing discussion suggests at once the types of cases in which an X-ray study of the optic canals should be undertaken as a routine procedure. It may be stated at the outset that whoever expects to find by this means a solution of his clinical puzzle in every case will be hopelessly disappointed. A negative finding here as elsewhere is of value. A negative Wassermann reaction rules out only *one* condition from the diagnostic possibilities, if it rules out that. On the other hand a negative optic canal Roentgenogram in a case of optic

nerve involvement rules out skull deformity, chronic inflammation of the walls, constriction, hyperostoses, fracture.

An optic canal Roentgenogram is therefore indicated in the following groups of cases:

1. Optic neuritis, choked disc, retrobulbar neuritis, optic nerve atrophy, etc.
2. Sphenoidal disease with or without optic nerve symptoms.
3. Orbital, ocular or optic nerve tumors.
4. Known or suspected fracture of the skull, whether recent or healed.
5. Foreign body in the orbit.
6. Skull deformities with visual disturbances.
7. Tumors of anterior or middle fossa.
8. Certain general diseases like Paget's, acromegaly, rickets.
9. Cases of tuberculosis with optic nerve symptoms.
10. Medico-legal cases, where compensation is claimed for loss of sight alleged to have been caused by injury.

In cases of optic neuritis the optic canal Roentgenogram may be expected to answer one or more of the following questions:

What is the underlying cause?

Is the canal large enough to allow the optic nerve to withstand the attack, or is it so narrow that the swelling optic nerve may be expected to atrophy from pressure?

Is there an associated periostitis of the optic canal walls (chronic)?

Is there extension of pneumatic spaces into the walls of the optic canals, and if so, to what degree?

Cases of so-called retrobulbar neuritis frequently turn out upon examination with the X-rays to be cases of tumors of hypophysis without any but visual symptoms, or tuberculosis of the sphenoid, etc. The diagnosis of retrobulbar neuritis is therefore unjustified in any case until these conditions are excluded by a proper X-ray examination.

In optic nerve atrophy we undertake our X-ray study in order to see whether the condition is caused by, or associated with, a constriction of the optic canal. If a constriction is present, is it due to skull deformity, inflammation, tumor, etc.?

Even in cases in which the atrophy of the optic nerve is a logical or expected finding fully, in accord with the clinical history or course, the optic canal Roentgenogram is of value. Thus, for instance, in a case of optic atrophy with a positive Wassermann reaction we have been able to establish that there had been a chronic periosteitis of the optic canal on one side with thickening of the wall but without constriction. (Case No. 3.) A small area of the density of bone at the center of the lumen suggested a degenerative process. The detailed history of this case and the course are given below.

In sphenoidal diseases without optic nerve symptoms an X-ray study will establish the degree of pneumatization of the optic canal walls and the size of the optic canal lumen. It will thus enable us to judge of the likelihood of involvement of the optic nerve and the probable effect such involvement would have upon it. In cases with optic nerve symptoms the points to look for are the same as mentioned under optic neuritis and optic nerve atrophy.

In cases of orbital, ocular and optic nerve tumors we can tell by means of the X-ray examination to what extent the optic canal is involved. Is it needless to say just how the Roentgenogram of the optic canal is of value in the diagnosis of fractures of the walls of the optic canal. An optic canal Roentgenogram should be taken in every case of fracture of or injury to the skull, to establish whether a fracture of the optic canal wall is present.

In cases of large foreign bodies in the orbit it is important to determine to what extent the optic canal became involved from the injury and how near the optic canal the foreign body is located.

The optic canal in skull deformities with visual disturbances will be the subject of a later article. The changes in the shape and size of the optic canals in oxycephaly and scaphocephaly have been mentioned. Changes are also found in plagiocephaly (oblique skull), microcephaly, hydrocephaly, etc.

Tumors of the anterior or middle fossa may extend to the optic canal and produce there destruction or constriction. These have been described in detail above (see Fig. 6).

In general diseases, like acromegaly, Paget's disease

rickets, there are frequently changes in the optic canals associated with the other changes in the bones of the skull. The changes in the optic canals produce visual disturbances.

As tuberculosis of the sphenoid is not uncommon in cases of pulmonary tuberculosis the appearance of visual symptoms, even when explained by the ophthalmoscopic picture, should prompt us to undertake an X-ray examination of the optic canals and the sphenoid bone in general.

That an optic canal Roentgenogram has not been required by the defense in every case of alleged loss of sight in which there had been no injury to the eyeball and in which optic nerve atrophy is the only ophthalmoscopic finding, can only be explained by the fact that the Roentgenography of the optic canals is relatively unknown to the profession. It has been an area of speculation almost like the glands of internal secretion. As soon as the legal profession becomes aware of the ease with which optic canal Roentgenograms can be obtained in each case and the important evidence they furnish, no case of alleged optic nerve injury will be tried without such Roentgenograms. They will be offered or demanded by either one or the other party in the suit. The demonstration in such cases of a long standing chronic process preceding the injury or a congenital condition like oxycephaly would be greatly against the plaintiff. On the other hand an optic canal Roentgenogram may definitely establish an injury to the optic nerve before any evidence is found in the ophthalmoscopic picture.

The ten types of cases enumerated are types in which the Roentgenography of the optic canal has already proven its value. A further use that may be suggested is for studying the mechanism in the production of atrophy of the optic nerve occuring in syphilis, multiple sclerosis, skull deformity, etc. Incidentally the position required for obtaining a Roentgenogram of the optic canal is superior to many others for studying the margin of the orbit and the closely adjacent portions of the facial bones. The case histories which follow illustrate nearly all of the types of cases in which optic canal Roentgenography is of value.

CASE NO. 1.—S. B., age 23, came to the Second Eye Clinic of the University of Vienna with the following his-

tory: She has always been well except for frequent colds in the head. Sixteen months ago she began to have severe headaches for which she was operated (septum and right ethmoid). At that time blindness in the right eye was discovered. At the present time she complains of pain in the left eye and its environment and impaired vision in the left eye (Dr. Guist).

She was referred to us for an X-ray examination (Central Roentgen Institute of the General Hospital of Vienna), and the following condition was found:

X-RAY REPORT.

The postero-anterior view shows in the region of the left orbital roof, corresponding to its posterior part which is made up of the processus horizontalis of the frontal bone and the lesser wing of the sphenoid, a remarkable change:

The crista alaris is clearly defined, but below it is a semicircular contour with sharp margins which demarcates a shadow about 2 centimeters wide and 1.5 centimeters long. We are dealing apparently with destruction of the bone by a tumor lying in the base of the left frontal bone.

Of interest is a small shadow bone projected immediately above the lesser wing of the right sphenoid. It is about six millimeters in diameter, of the intensity of bone. Possibly we are dealing with another tumor which is causing the visual disturbance in the left eye.

We had the patient return for another X-ray examination. At this time Dr. Mayer took Roentgenograms of the optic canals by the method described by the author, and found the following condition.

The optic canal of the left side is large, somewhat circular in cross section, surrounded by a thin wall of bone which is denser in the lower than in the upper half. Its calculated "A" diameter is 4.9*mm* and the calculated "B" diameter is 4.9*mm*.

The right optic canal (see Fig. 3) differs in shape, size and structure of its wall from that of the left side. It is triangular in cross section, smaller in its lumen, the calculated "A" diameter being 3.6*mm* and the calculated "B" diameter being only 3.1*mm*. The wall is thicker and denser than that of the right side. There are shadows of the intensity of bone immediately adjacent to and continuous with it above, mesially and laterally and in the lumen. The upper shadow is heart-shaped, of uniform intensity measuring about 6.0*mm* in each of its principal diameters. The mesial shadow is triangular, densest at its center; the sides of the

triangle average about 8.0*mm*. The external shadow is of uniform density, directly continuous with optic canal wall, triangular, each side measuring about 2.0*mm*.

It is evident that the tumor of the left side has constricted the optic canal and is responsible for the blindness of this eye.

CASE No. 2.—Patient, 51 years old, with marked oxycephaly. Blind since the age of four, has no light perception in either eye. X-ray examination shows small, constricted optic canals of the shape characteristic of the skull deformity (somewhat triangular).

CASE No. 3.—A. J., age 35. Clinically optic atrophy left and optic neuritis right. Wassermann four plus; X-ray examination shows: right optic canal normal. Left optic canal has very thick dense walls (see Fig. 4) and a dense shadow within the lumen. The wall thickening indicates an old periostitis of the optic canal. The calcification within the lumen indicates calcification of the optic nerve, making the prognosis for improvement of sight in this eye hopeless.

After antisyphilitic treatment the vision in the right eye returned to almost normal. No improvement in the left eye.

CASE No. 4.—H. G., age 12 years. Clinical diagnosis: Optic atrophy, right, simple.

Patient first noticed blindness of right eye two months before he presented himself at the clinic. Vision: right, hand movement at a distance of one foot; left $\frac{2}{3}\frac{0}{0}$. Right pupil immobile. Right disc slightly pale. Wassermann negative. The atrophy was ascribed to meningitis which the patient had in childhood.

X-ray examination shows: Left optic canal normal. Right optic canal triangular in shape, particularly flat at the roof. Evidence of increased intracranial pressure in frontal and parietal regions. Careful study of the sella turcica shows destruction of the right posterior clinoid process with a sharp-pointed remaining stub. The point is directed backward and upward. The upper outline of this stub sweeps downward and forward from this point below the level of the sella outline, across the middle fossa to its anterior wall. The apparently normal though hazy sella outline seen above the line just described corresponds to the left side of the sella turcica.

An X-ray diagnosis of right sided suprasellar erosion due to a tumor, was made following which careful plotting of the

field showed: Right—hand movement in upper nasal field only. Left—temporal hemianopsia. The clinical diagnosis of tumor cerebri, right, was now made.

CASE No. 5.—E. B. Clinically secondary optic atrophy, left.

X-ray examination shows (Fig. 5) right optic canal normal. Left optic canal constricted, its lower outer wall thickened and a spur extending from it to the center of the lumen. The adjacent anterior clinoid process is pneumatized, its walls thickened. The thickening and exostosis into the optic canal are due to chronic inflammation within this clinoid process.

CASE No. 6.—H. P., male. Recent impairment of vision right. Secondary optic atrophy left. X-ray examination shows: Right, optic canal normal. Left, optic canal constricted to 2.0*mm;* its walls thick and irregular; the adjoining anterior clinoid process pneumatized and its walls thickened. No evidence of hyperostoses elsewhere in the skull. This is a constriction due to chronic sphenoiditis.

CASE No. 7.—Miss C. L., 34. Headaches and dizziness for the past 10 years; choked disc 10 years ago. Was then treated for sinusitis. X-ray examination then and seven years later said to be negative. Now has bilateral optic atrophy, and difficulty in maintaining her balance.

X-ray examination shows an almost total destruction of the body of the sphenoid, so that one cannot tell whether the lesion was originally intra- or extrasellar. The roofs of both optic canals are destroyed (Fig. 6), indicating extension into the anterior fossa. On the right side there is evidence of stasis in the jugular fossa. The apices of the petrous portions of both temporal bones are eroded, the erosion on the right side extending as far as the semi-circular canals and breaking into the superior semi-circular canal. X-ray diagnosis: Tumor of middle fossa extending into anterior and posterior fossæ.

CASE No. 8.—H. W., female, age 5½ years. Optic nerve tumor, right, operated 2 years ago by Dr. Arnold Knapp.

X-ray examination shows: Right optic canal a little larger than the maximum normal found in our measurements, being 5.6 × 6.0*mm* (maximum 5.6 × 5.5*mm*). Left optic canal shows marked enlargement. The calculated dimensions of its cross section are 7.0×8.0*mm*. This represents an enlargement, compared with the left side, of 25% in one diameter and 45.5% in the other diameter.

CASE NO. 9.—A. W. Optic nerve tumor, left; operated by Dr. Arnold Knapp 3 weeks ago.

X-ray examination (Fig. 7) shows marked enlargement of the left optic canal, slight enlargement and haziness of the left sphenoidal fissure. There is no evident change in the left anterior clinoid process. The calculated diameters of the optic canals are: Right, 4.3 × 5.2*mm;* left, 7.5 × 8.0*mm*. This represents an enlargement of the left optic canal, as compared with the right, of 71% in one diameter and 54% in the other diameter.

CASE NO. 10.—J. D., age 34, male. Was struck on the head 3 weeks ago. Was unconscious 2 days. Now has total ophthalmoplegia, left.

X-ray examination of the optic canals (see Fig. 8) shows: The right canal is normal in shape, size and structure of its walls. The left optic canal is normal in size. At its lower outer portion is a fine fracture line extending from the sphenoidal fissure into the optic canal. The lower fragment is displaced downward, toward the sphenoidal fissure.

CASE NO. 11.—J. K. Fell down a flight of stairs 5 months ago. Claims to have lost the sight of his left eye. Now has simple optic atrophy.

X-ray examination shows a markedly deformed, oxycephalic skull, with extremely thin frontal and parietal bones and evidence of long standing increased intracranial pressure. The sella turcica is large and deep, its anteroposterior diameter being 1.5*cm* and its depth being 1.7*cm*.

The optic canals have the shape characteristic of the skull deformity, being oblong and triangular. The left optic canal is constricted in the "A" diameter which measures only 2.8*mm*. Its "B" diameter is 4.4*mm*. The right optic canal measures 4.1 x 5.4*mm*.

It is evident that we are dealing with a case of optic atrophy associated with oxcephaly, which therefore dates back to the patient's infancy. As the patient is suing the landlord for a huge sum of money, the value of the X-ray examination, particularly of the optic canal Roentgenograms, to the defense is obvious. The ophthalmologist who examined this patient later for the defense missed the skull deformity and did not recognize the need of optic canal Roentgenograms.

CASE NO. 12.—Mrs. G. S., age 55; clinically retinitis pigmentosa.

X-ray examination shows a curved shadow of the intensity of bone corresponding in position to the cavernous por-

tion of the internal carotid artery. On the optic canal Roentgenograms an annular shadow is seen immediately temporal to the shadow of the optic canal. It is a little smaller than the optic canal. This is a bilateral calcification of the internal carotids.

CASE NO. 13.—J. E., age 32. Optic atrophy with ring scotoma and retinitis pigmentosa.

X-ray examination shows plagiocephaly due to obliteration of the left side of the coronal suture.

The optic canals are both deformed, the right being triangular and characteristic of the skull deformity, the left being irregularly trapezoidal due to excessive pneumatization of the left side of the sphenoid, the canal being almost entirely surrounded by pneumatized bone.

CASE NO. 14.—R. B. Impaired vision and exophthalmos of left eye 3 years. Optic nerve atrophy. Clinical diagnosis: optic nerve tumor?

X-ray examination shows: Small wedge-shaped erosion in the outer wall of the right optic canal, hyperostosis of the lower inner wall of this canal and of the mesial part of the lesser wing of the left sphenoid, immediately adjacent to the optic canal. This combination of fine erosions and osteomata is characteristic of endotheliomata and we are probably dealing with an endothelioma of the optic nerve sheath.

CASE NO. 15.—I. I., age 35. Optic atrophy with bitemporal hemianopsia. Impairment of vision began two years ago in left eye. Right eye involved later.

VISION O.D. $\frac{8}{10}$; O.S. hand movement in nasal field. A—R pupils. Had some nose operation and salvarsan treatment for his eye trouble 2 years ago. Now complains also of tinnitus in left ear, which began 6 months ago, worse at night. Continuous frontal headaches and itching sensation over vertex of head.

X-ray examination: Right optic canal normal in shape, size and structure of its walls. Lower temporal part of wall of left canal flat. Immediately below this part of wall is a small calcified mass. The Roentgenogram of the sella turcica shows a small bubble-shaped mass overlapping the shadow of the anterior sella wall, situated partly within the fossa and partly within the sphenoidal sinus in front of it. It has a very thin calcified wall. Oblique projection, to eliminate the possibility of this being a cell within the temporal bone, shows this mass to be situated under the left anterior clinoid process and eroding the under surface of

the latter. An X-ray diagnosis of cyst of the hypophysis was made. We considered this an ideal case for the Hirsch operation as the cyst should be easily reached through the left sphenoid. The patient was referred to a surgeon who, however, entered through the right temporal region and found some inflammatory tissue but no cyst. Another X-ray examination undertaken 3 months later shows the original lesion still present but larger. Roentgenograms of the profiles of the optic canals taken six weeks later confirm the presence and situation of the lesion.

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CONCERNING THE OCULAR SYMPTOMS OF ERYTHREMIA (CHRONIC POLYCYTHEMIA VERA), WITH SPECIAL REFERENCE TO THE FUNDUS PICTURE.[1]

By Drs. G. E. de SCHWEINITZ, Philadelphia, Pa., and ALAN C. WOODS, Baltimore, Md. (By invitation).

(With one illustration in colors on Text-Plate III.)

THE object of the present communication is to describe the usual fundus picture in erythremia (polycythemia vera) as determined by an examination of the records of patients kindly placed at our disposal by the medical staff of Johns Hopkins Hospital and of the Hospital of the University of Pennsylvania.

Only such case histories have been utilized, thirty in number, which contain reliable notes as to the ocular examinations, as well as to the medical investigations, the ophthalmoscopic studies in all instances having been made by those who are competent in this respect.

Although the literature which pertains to this subject is voluminous and comprises a notable number of papers which contain the records of many cases and series of cases, either personally reported or gathered from previous publications, there is none, in so far as we have been able to ascertain, which is devoted solely to the purpose of determining the usual fundus picture, as this is revealed by a goodly number of patients submitted to such ocular investigations as those we are able to present for your consideration.

The usual ocular vascular changes in polycythemia vera

[1] Read before the American Ophthalmological Society, May, 1925.

are well known, especially those which are found in the retinal vessel distribution, and have been frequently pictured and described, but not exactly as we hope to portray them in relation to the erythrocyte count and the hemoglobin percentage.

We are not, therefore, concerned with the large literature of this subject in the sense of an elaborate record and analysis, which have often been made, and any one interested will find plenty of material in this respect, for instance in the papers of Turk (1) in Germany, of Engelbach and Brown (2), of Edward Jackson (3), of T. B. Holloway (4), and of W. S. Lucas (5) (a comprehensive review of 189 cases, 149 of which were unquestionably polycythemia vera) in this country; in the Gale Lecture by Watson (6), in Foster Moore's (7) book on *Medical Ophthalmology*, and in F. Parker Weber's (8) admirable monograph, with an extensive bibliography, on "Polycythemia, Erythrocytosis and Erythremia," in England.

We were led to introduce this subject partly for the reason stated, partly as a small supplement to Thomas B. Futcher's (9) recent excellent essay *Clinical Aspects of Polycythemia*, based on a detailed medical study of the case histories of patients with erythremia treated in Johns Hopkins Hospital, and partly because some of the reports in ophthalmic literature on the fundus changes in polycythemia dwell upon conditions or complications which are unusual as compared with ordinary manifestations. Thus, one Continental author very accurately described what he entitles the "Fundus polycythæmicus," namely, notable retinal vascular changes, not to the exclusion, however, of choked disc, which, as he points out, has been described, but which he has not observed. In his cases, he states, as long as they were under observation, neither prominence of the nerve-heads, nor enlargement of the blind spots, nor hemorrhage, nor visual disturbance was noted. On the other hand, a British authority dwells especially upon the ophthalmoscopic changes which "may be extreme in degree," and upon "the retinal hemorrhages which may be very profuse."

Fully recognizing that "extreme" eyeground alterations and gross visual disturbances may occur, our object is to define the usual, and in a sense pathognomonic, ophthalmo-

The usual fundus picture in polycythemia vera.

scopic indications of this disease, that is, to adopt Ascher's (10) phraseology, the "fundus polycythæmicus."

The following condensed historical note may serve as an introduction to the discussion which follows:

When Vaquez (11), in 1892, reported a case of cyanosis with polycythemia, the red cells and hemoglobin being greatly increased, he thought the condition was due to congenital heart disease, but autopsy three years later revealed the diagnostic error. During the subsequent ten years a number of cases of chronic cyanosis with polycythemia were published, for example, by Richard Cabot (12) and by McKeen (13) in our country, by Rendu and Widal (14) in France, and by Cominotti (14½) in Germany. But this pathologic manifestation was first referred to as a clinical entity, as is pointed out by Osler (15) in his well-known paper, by Saundby and Russell (16) in England, in 1902. It was not, to quote W. S. Lucas, "until the appearance of Osler's communications in 1903 and 1904, that this condition was brought prominently before the profession of this country, and its existence established." Hence it is often called Vaquez-Osler disease, but more appropriately, considering its pathogenesis, erythremia, a term introduced by Turk.

Although the ophthalmoscopic changes incident to this disease were recorded by Richard Cabot (17) in Boston in 1900 as "dark dilated veins, arteries contracted" (examination by Dr. Amadon), by Stockton (quoted by Osler) in Buffalo in 1903, as "hyperemic discs, vessels and particularly veins engorged and tortuous," and by Kōster (18) in 1906 as those of venous hyperemia, the first satisfactory description of the fundus picture in erythremia, illustrated by an excellent water color, was made by Uhthoff (19) in 1906, before the Heidelberg Ophthalmological Society. He found as salient features: unevenly dilated and dark-colored retinal veins; arteries only very slightly wider and darker than normal. Hemorrhages and exudation were absent, and there was no disturbance of direct or indirect vision.

One year later (1907) Edward Jackson (20) published a description of the ophthalmoscopic appearances in chronic cyanotic polycythemia as he had studied them in a Jewish woman aged sixty, in 1902 and 1903, and illustrated his paper with a water color which resembles the one by Uhthoff, save only that he depicts a few small retinal hemorrhages of roundish form, the first, so far as he was aware, which had been reported to date.

In the same year (November, 1907) one of us (G. E. de S., 21) reported a typical case of chronic polycythemia vera before the Ophthalmic Section of the College of Physicians of Philadelphia, also illustrated by a water color of the fundus, which is almost a counterpart of the one which Uhthoff utilized in his article, and which portrays, as his does, the usual fundus picture in erythremia which may be regarded as its pathologic norm. (See Plate.)

A brief reference to the important departures from the usual ophthalmoscopic appearances may be summarized thus: Moderate blurring of the nerve-head and surrounding retina has been observed, but a choked disc, ophthalmoscopically and microscopically in no wise differing from that produced by increased intracranial tension, was first observed by Carl Behr (22), and was attributed by him to local edema of the papilla and end of the optic nerve. In our country, W. T. Shoemaker (23) made a similar observation, the patient having been studied from the general standpoint by W. S. Lucas. Shoemaker states that the changes were those of "typical inflammatory choked disc, identical with those frequently seen with intracranial disturbance." In one of the cases in R. Foster Moore's series "the disc changes differed little from the papilledema of brain tumor."

Other important ophthalmoscopic changes are concerned with retinal hemorrhages; only a few may be present, as noted in Jackson's report, and as they have been recorded by Walter Parker and Slocum (24), and a number of other observers. Rarely they may be extreme in degree and accentuation, and in one of Foster Moore's patients "the condition simulated thrombosis of the central retinal vein so closely, that one was led to suggest that prominent cerebral symptoms, which were present, might be due to thrombosis of the cranial sinuses, as indeed, proved to be the case at the autopsy."

Visual disturbances in polycythemia vera are not infrequent, and have been noted by many observers, eight times in a series of ten cases reported by Christian. They are well discussed by Harry Friedenwald (25). They vary from blurred vision to complete blindness, in the last instance sometimes without evident ophthalmoscopic changes to account for it.

They also include muscæ, scintillating scotomas, temporary hemianopsia and ophthalmic migraine. In a case studied by one of us (G. E. de S.) typical migraine was a persistent symptom long before the general examinations revealed erythremia.

Visual impairment has been attributed to retrobulbar and

papillary neuritis (Friedenwald), and to pressure of an engorged central vein on the optic fibers (E. Jackson), and to intracranial lesions. Diplopia and nystagmus have been noted.

ANALYSIS OF THE MATERIAL.

Of the 30 patients whose clinical histories have been utilized in this report and placed at our disposal, as recorded in the first paragraph of this paper, practically all of them were undergoing active treatment, and hence the blood picture was constantly changing, the erythrocyte count and hemoglobin percentage usually changing. The blood counts and hemoglobin percentages recorded in the tables are those which synchronize most nearly with the date on which the ophthalmoscopic examinations were made. This accounts for the low figures which occasionally appear.

I. *The Fundus Picture in Polycythemia Vera.*—The ophthalmoscopic appearances observed in this series of polycythemia patients may be classified into four general groups. The first group of nine patients comprises those in whom the eyegrounds appeared to be normal, or at least were not distinctive. The second group of eight patients includes those in whom the only abnormal finding was dilatation of the veins, without notable alteration of color. The third group of ten patients consists of those in whom dilatation and tortuosity of the veins, together with definite changes in the color of these vessels and of the fundi, were specifically recorded, and no other anomalies were observed. The fourth group of three patients comprehends those patients in whom, in addition to changes in the size, caliber, and color of the retinal veins, the presence of hemorrhages or exudates was noted. It is noteworthy that in 27 of the records there is a definite statement that "no hemorrhages or exudates" were present. In the eyegrounds of three patients only hemorrhages or exudates were discovered. All of these patients had a high hypertension with arteriosclerosis, and in each the retinitis was recognized and noted as an arteriosclerotic process.

Fourteen of the patients in this series revealed a definite hypertension. Effort was made to correlate this hypertension

with the fundus changes, but this could not be done inasmuch as the hypertension cases pertain irregularly to the three main groups which have been arranged. Likewise, several patients were the subjects of nephritis, myocardial insufficiency, vascular thrombosis, and other vascular or more remote disturbances, but no connection could be traced between these allied conditions and the ophthalmoscopic picture, except in the patients with arteriosclerotic retinitis. It was not until the blood picture was studied in relationship to the ophthalmoscopic picture that any apparent connection between the two was discovered.

TABLE I.—POLYCYTHEMIA VERA; EYEGROUNDS NORMAL AND NOT DISTINCTIVE.

Hosp. No.	*Blood Pressure*	*Nephritis*	*Hb. Per Cent.*	*R. B. C.*	*Fundi*
20,865	Systolic 145	None	90	6,688,000	Fundi show no abnormalities.
35,565	145/90	None	102	6,500,000	Fundi neg. (disp.)
35,926	210/100	None	108	6,660,000	"Arteries small, no hemorrhages or exudates."
35,821	135/95	Chronic nephritis	112	6,640,000	"Eyegrounds normal."
41,364	120/75	None	105	7,500,000	"Eyegrounds essentially normal."
50,581	115/70	None	105	6,296,000	"Tint of discs and retinas normal."
51,395	180/120	None	95	5,256,000	"Slight vascular sclerosis, no hemorrhages."
51,793	240/135	None	114	8,280,000	"Retinal veins normal size. No hemorrhages."
U.P.H.		None	130	6,200,000	No abnormal distention of veins; no hemorrhages.

II. *Relationship of Blood and Ophthalmoscopic Pictures.*—The notable change in the blood picture of polycythemia is an increase in the erythrocytes and the hemoglobin percentage. It would be expected, then, that any fundus changes characteristic of polycythemia would vary in intensity as the erythrocyte count and hemoglobin percentage rose. An ex-

amination of Tables I, II, and III indicates that, although in general terms this is true with respect to the hemoglobin percentage, it is not so evident in relation to the erythrocyte count.

TABLE II.—POLYCYTHEMIA. CHANGES IN VESSELS ONLY FUNDUS ABNORMALITY NOTED.

Hosp. No.	*Blood Pressure*	*Nephritis*	*Hb. Per Cent.*	*R. B. C.*	*Fundi*
30,317	Systolic 135	None	125	8,200,000	"Marked engorgement of veins. No hemorrhages or exudates."
33,039	Systolic 120	None	117	6,632,000	"Veins fuller and darker. No hemorrhages or exudates."
38,655	130/108	None	105	6,032,000	"Normal except for dilated veins. No hemorrhage."
46,397	150/70	Chronic nephritis	124	7,888,000	"Veins very full and dark color. No hemorrhages or exudates."
47,899	126/90	None	110	6,650,000	"Veins full, arteries normal. No hemorrhages or exudates."
48,854	170/110	None	112	7,150,000	"Veins full and engorged. No hemorrhages or exudates."
E.B.V.A	200/130	None	114	9,640,000	"Dilated veins, no change in color of fundi. No hemorrhages or exudates."
J.B.L.		Chronic nephritis	125	9,200,000	Dark dilated veins; arteries unchanged; no hemorrhages or exudates.

Table I shows the findings in the patients where the fundi were noted as normal, or at least as not distinctive. The erythrocyte count in these nine patients varies from 5,256,000 to 8,280,000, an average of 6,768,000, and the hemoglobin percentage varies from 90 per cent. to 114 per cent., an average of 104 per cent. In six of these nine patients, however, the hemoglobin is below 110 per cent.

TABLE III.—POLYCYTHEMIA. ENGORGED VEINS AND CHANGES IN COLOR OF FUNDI ONLY CHANGES.

Hosp. No.	*Blood Pressure*	*Nephritis*	*Hb. Per Cent.*	*R. B. C.*	*Fundi*
38,628	200/140	None	135	9,104,000	"Veins full, fundi congested, etc. No hemorrhages."
40,792	130/80	Chronic nephritis	125	7,520,000	"Fullness of veins and very dark color of fundi. No hemorrhages or exudates."
39,939	175/110	None	155	10,724,000	"Slight haziness of discs, tremendous engorgement of veins. Great congestion. No hemorrhages or exudate."
40,957	142/84	Chronic nephritis	115	6,400,000	"Fundi dark colored. Veins full and dark colored."
41,132	140/90	None	150	9,152,000	"Eyegrounds very dusky in color. Veins much engorged and purple in color. No hemorrhages or exudates."
49,365	155/110	None	133	8,680,000	"Marked deepening of red color of fundi. Veins almost black. No hemorrhages."
50,736	155/80	None	138	8,912,000	"Veins are full, very dark red, almost black. Fundi deep red. No hemorrhages."
52,186	160/100	None	125	10,912,000	"Veins full, tortuous, almost black. Fundi oculi more intense than normal."
U.P.H.		None	120	9,250,000	"Veins greatly enlarged, dark and tortuous; arteries unchanged; eye-ground darker than normal."
U.P.H.		None	170	12,900,000	"Veins distended, not tortuous, fundus darker than normal."

Table II portrays the findings in patients where it was definitely noted that a dilatation of the veins was present. Here the erythrocyte count runs between 6,032,000 and 9,640,000, an average of 7,836,000, that is, a higher level than was found in the patients with practically normal fundi. The hemoglobin percentage, however, varies between 105 per cent. and 125 per cent., an average of 115 per cent. In seven of the eight patients the hemoglobin was 110 per cent. or more.

Table III exhibits the results in those patients where, in addition to the dilatation of the veins, definite alterations in the color of the fundi were noted. In these patients the erythrocyte count varied between 6,400,000 and 10,912,000, an average of 8,656,000, a definitely higher level than in the first two groups of patients. The hemoglobin percentage varied from 115 to 155, an average of 135. In nine of the ten patients in this group the hemoglobin was 125 per cent. or more.

A study, therefore, of all these patients indicates that the usual fundus picture of polycythemia, if there is a characteristic fundus picture, consists in dilatation, sometimes uneven, of the retinal veins, and deepening of their color, and in alterations, cyanotic in type, of the normal color of the eyegrounds without hemorrhages, exudates, or changes in the optic nerve, and generally without marked change in the caliber and tint of the arteries. The relationship of these changes to the blood picture appears to be as follows: There is no striking difference in the erythrocyte count of those polycythemia patients who have ophthalmoscopically normal or, at least, not distinctive fundi and those who show venous dilatation, although the figures in the second are higher than those in the first group. There is, however, a more striking difference in the hemoglobin percentage, the venous dilatation occurring rather regularly when the hemoglobin passes 110 per cent.

The cyanosis of the retina appears only when the polycythemia becomes more severe, the erythrocyte count usually being at least in the neighborhood of 7,000,000,[1] and the hemoglobin 125 per cent. or more. The change in color of the fundus appears to be due more to an increase in the

[1] In all except one case it was above 7,000,000.

hemoglobin percentage than to an increase in the erythrocyte count. In the series as a whole, there is only one patient with a hemoglobin of 125 per cent., where it was not specifically noted that the eyegrounds exhibited alterations in color, while there are four patients in whom the erythrocyte count was in the neighborhood of 8,000,000 or more, and in whom no notation was made of alteration in the color of the eyegrounds. It is, however, almost impossible with exactness to separate these two factors, hemoglobin percentage and erythrocyte count, which in the main increase together. So far as this can be done, however, the cyanosis of the retina appears to accompany the increase in the hemoglobin percentage, as might be expected.

These fundus changes, dilatation of the veins and cyanosis, are essentially the same as those which accompany the disease in other parts of the body: Dilatation of the venules of the face, ears, mucous membranes of the lips, conjunctiva, etc., with the characteristic dusky color. Uhthoff supposed the dilatation of the veins, especially in the retina, to be due to an atony of the vessel wall. Minot and Buckman have shown that the erythrocytes in polycythemia are sometimes more resistant and sometimes less resistant than normal, and that the serum of polycythemic patients may at times be hemolytic. It is possible that such changes in the erythrocytes and blood serum, or some unknown toxic substance liberated by the hyperplasia of the erythroblastic bone marrow, may have a direct action on the vessel wall, or may have a depressor effect on the vasomotor autonomics, and so be responsible for the dilatation of the veins. On the other hand, as noted by Futcher in his general studies of polycythemia, it has been shown there is an increase in the total volume of the blood due almost entirely to the increase in the bulk of the red cells. Such increase has been reported to be as great as three times the normal. It seems more probable that the retinal venous dilatation so commonly observed is merely the yielding of the thinner venous wall to the increase in blood volume. The alteration in the color of the retina, cyanotic in type, is exactly the change that would be expected, and must necessarily accompany any such overloading of the venous circulation and great increase in the hemoglobin percentage.

III. *Influence of the General Complications of Polycythemia on the Fundus Picture.*—A. *Hypertension.*—Hypertension was a frequent, but by no means constant, complication observed in these patients. It was evident in 15 of the 30 patients here reported. Three of these patients showed a definite arteriosclerotic retinitis (Table IV), in each instance observed personally by one of us (A. W.). While these patients had polycythemia, with characteristic dilatation of the veins, yet the flame-shaped hemorrhages and cotton-fluff exudates were typically those found in arteriosclerotic retinitis, and there were present the usual changes in the vessels associated with retinal angiosclerosis. There is no evidence that retinitis in these patients was dependent upon the underlying polycythemia.

B. *Hemorrhages.*—There is a definite tendency to hemorrhage by patients with polycythemia. Thus Futcher, whose clinical study includes the majority of the patients here reported, notes that one patient of the series had severe hemoptysis without clinical signs of tuberculosis, one patient severe gastric hemorrhage, one patient repeated hematuria, and in two patients red blood cells were found in the urine upon microscopic examination. Evidently retinal or choroidal hemorrhages may occur in polycythemia, without vascular sclerosis, as has previously been described. Such hemorrhages should not be regarded as a characteristic change in polycythemia, for it is plain from this study that they are of infrequent occurrence, certainly uncommonly recorded. It is possible that small inconsistant retinal hemorrhages are more frequent than the records reveal. The one Edward Jackson first found in his patient escaped later detection until he dilated the pupil. He suggests that the extreme viscosity of the blood prevents the frequent occurrence of hemorrhages. The gross hemorrhagic changes which Foster Moore observed in one of his patients, presenting an appearance indicative of thrombosis of the central retinal veins, must be most unusual; he also found on microscopic examination great infiltration of the choroid coats with blood.

C. *Other Vascular Lesions.*—There is a very definite tendency of polycythemia patients to develop vascular complications. Futcher, in his clinical study of 32 patients (which

TABLE IV.—POLYCYTHEMIA. HEMORRHAGES AND EXUDATES NOTED IN ADDITION TO USUAL FINDINGS

Hosp. No.	*Blood Pressure*	*Nephritis*	*Hb. Per Cent.*	*R. B. C.*	*Fundi*
33,697	245/140	Chronic nephritis arterio-sclerosis	105	5,932,000	"Blurring of neuro-retinal outlines, edema of retina, small hemorrhages below discs, pale exudates." Condition improved, and it is noted that picture in fundi cleared except for overfilled veins.
44,181 etc.	210/130	Chronic nephritis	112	7,712,000	Definite picture of arterio-sclerotic retinitis described in early histories, with hemorrhages and exudates. Under treatment for hypertension this cleared, and it was noted "fundi essentially negative except for site of old hemorrhages and exudates." Patient returned two months later, with polycythemia, and it was noted "arteries definitely sclerosed, veins distended."
G.W.	190/90	Chronic nephritis	110	7,200,000	Widely distended, definite arteriosclerotic changes in vessels and at arterio-venous crossings, slight haziness of neuroretinal outlines, and numerous deep cotton-fluff exudates. Picture that of arteriosclerotic retinitis, with great distention of veins.

includes the majority of our patients), reports six patients with a hemiplegia, which, owing to the slowness of its evolution, was supposed to be due to cerebral thrombosis, in several instances confirmed at autopsy. Two other patients had varicose veins of both legs, suggesting a venous thrombosis; two patients had varicocele; and one patient developed an acute phlebitis without fever. In view of this marked tendency to venous thrombosis, it seems quite probable that the typical choked disc, reported by various observers in polycythemia, may be due not to the mechanism which Behr suggested, but to a cerebral venous thrombosis. In only one patient of this series were any noteworthy changes found in the papilla, and they were limited to haziness of the neuroretinal outlines, exactly as has been reported by other observers, for example, by Stockton.

D. *Conjunctival Vessels.*—A common and well-known manifestation in erythremia is a marked and often diffuse conjunctival hyperemia of the conjunctiva in the sense of dark-colored, injected, and dilated vessels, unassociated with inflammatory symptoms and abnormal secretion. This may antedate the retinal vascular changes, or be conspicuous when they are not present, or are not pronounced. Sometimes the sclera has a distinctly bluish tint.

Such conjunctival hyperemia was a notable feature in two of the patients in our series, and in one of them was present for some years prior to the discovery of its real cause.

Slit-lamp examination of the eyes of one of these patients (only one investigation was possible) emphasized the dark purplish color of the conjunctival veins and the bluish tint of the sclera, but did not reveal any changes in the vessel walls. No alterations in the iris vessels were found. Further investigation with the aid of this instrument is indicated.

We desire to express our thanks for the privilege of examining the records of polycythemia patients to Drs. Longcope and Futcher of the Johns Hopkins Hospital, and to Dr. Alfred Stengel, Dr. George Piersol, Dr. Joseph Sailer, and Dr. Samuel Leopold of the Hospital of the University of Pennsylvania.

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GLAUCOMA IN MYOPIC EYES.[1]

By Dr. ARNOLD KNAPP, New York.

WHILE the anatomic peculiarities of the hypermetropic eye predispose it to glaucoma, glaucoma of the simplex variety does not infrequently occur also in myopia. Lange (*K. M. A.*, 1920, v. 50, p. 540) and Gilbert (*Graefe's Archiv.*, v. 82, p. 393) believe that the refraction of the eye determines the clinical form of glaucoma. They found myopia present in about $\frac{1}{3}$ of the cases of glaucoma simplex while myopia occurred in only $\frac{1}{10}$ of the cases of congestive glaucoma.

I have the records of 32 cases of glaucoma in myopic eyes with the following findings. The degree of myopia in 26 was below 5 D; in 6 it was between 5 and 10 D. The age varied between 40–50 in 3; between 50–60 in 6; 60–70 in 11; over 70 in 12; the majority therefore was over 60, perhaps somewhat older than the rule. The type of glaucoma was always the chronic one (simplex); in one case only were there some symptoms of uncompensation (congestion), there were no cases with acute symptoms.

The features in these eyes were: Depth of the anterior chamber, in only 2 was the anterior chamber shallow, in 12 it was normal, and in 17 it was deep. The intraocular tension was usually but slightly elevated, in only 6 was it over 40 (Schiotz); it was generally around 32 (28 to 36). In 4 the tension was low 18, 18, 20, 20, constituting a group to which I will return later. The optic nerve changes consisted in a shallow cup with central depression of the vessels, the bending of the vessels occurring especially at the upper margin of the disc. In others there was a nasal displacement of the vessels,

[1] Read before Am. Ophth. Soc., Washington, May 4, 1825.

a well-developed conus, and in many a complete halo; the optic nerve showed usually distinct atrophy. Axenfeld (*D. O. G.*, 1924, p. 140) says that the value of Bjerrum's sign is not realized because of the myopic conus. I am not able to confirm this as our fields taken on a Bjerrum screen at 1 meter with a 1 or *2mm* object showed all varieties of glaucomatous field-defect from a prolongation of the blind spot up or down or both, joining to form a complete ring to a concentric contraction.

As for treatment, some were controlled by miotics, others were operated upon by iridectomy, cyclodialysis or some form of scleral resection. Though some cases went on slowly to progressive deterioration whether they were operated on or not, the results of the operative cases on the whole were surprisingly good, considering the degenerative nature of the lesion. The technic of the operation was simplified by the deep anterior chamber, and never did one encounter the terrific problems and disasters furnished by the highly hypermetropic eye with practically no anterior chamber and rudimentary cornea.

There were in addition 2 cases of glaucoma with myopia in adolescents aged 16 and 22. In one with myopia of 1.5D, $S=\frac{20}{100}$, tension 45, deep anterior chamber, peculiar iris atrophy, optic nerve, deep cup, central depression of vessels and halo. The field was concentrically contracted. The sclera at operation was found unusually thin. In the second case also with a myopia of 1.5D; $S=\frac{20}{50}$ (3 years ago vision was emmetropic), tension 36, superficial atrophy of iris, deep anterior chamber; optic disc, deep cup. Field, concentric contraction. Both of these cases showed trophic changes in the iris, and a definite thinning of the anterior scleral capsule which suggested that the myopia was secondary to the glaucomatous process.

Two cases deserve mention on account of the subnormal tension 18 and 20, notwithstanding cupping of the optic nerve-heads and definite glaucomatous field defects. Though the diagnosis of glaucoma in these cases is difficult, repeated tonometric and perimetric examinations will indicate the line of treatment.

In conclusion may I summarize my observations by saying

that glaucoma in myopic eyes is of the simplex type, with deep anterior chamber, low increased tension, characteristically shallow cup, and the usual glaucomatous field defects and that operation, if indicated, gives us on the whole suprisingly good results.

SEPARATION OF THE CHOROID, WITH REPORT OF A SPONTANEOUS CASE.[1]

By Drs. F. H. VERHOEFF and J. H. WAITE.

From the Massachusetts Eye and Ear Infirmary.

(With one illustration on Text-Plate IV.)

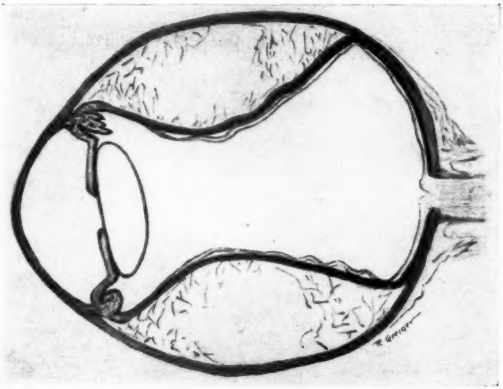
VON GRAEFE (1) in 1854 was the first to demonstrate clinically a separation of the choroid. Since then, separations of the choroid have been reported arising from tumor, from trauma, from intraocular operations, from nephritis, and from unknown causes.

Neglecting separations following tumor of the choroid, and passing to those following trauma, one may briefly summarize certain aspects of the cases reported by Michel (2), Mules (3), and Story (4). All three record a definite antecedent trauma. Michel states that the tension of the eye affected was reduced, while in the other two cases there is no record of tension. The site of the separation was macular in the cases of Michel and Story, and not given in the other case. None of the three case reports contain any statement concerning the depth of the anterior chamber.

Separations of the choroid following intraocular operations have been discussed by Fuchs (5), Meller (6), Barkan (7), Marshall (8), and others. It is now generally believed that such separations are due to hypotonus from wound leakage, and that they usually recede whenever normal intraocular pressure is restored. The hypotonus from old inflammatory conditions, and from contracting bands, is a cause for separation of the choroid, but here the condition is of little clinical importance.

[1] Read before the American Ophthalmological Society, May, 1925.

ILLUSTRATING ARTICLE OF DRS. F. H. VERHOEFF AND J. H. WAITE, "SEPARATION OF THE CHOROID, WITH REPORT OF A SPONTANEOUS CASE."



A Case of Separation of the Choroid

Separations of the choroid attending nephritis have been reported first in 1898 by Ewetzky (9), and later by Lauber and Adamuk (10). In Ewetzky's case, there was chronic interstitial nephritis, and the eye showed a tension which rose to equal 41 millimeters of mercury, and also a very shallow anterior chamber. Histological examination of the eye showed extensive vascular degeneration, with hemorrhage and transudate into the ocular tissues and beneath the choroid. In the case of Lauber and Adamuk, in which there is no statement of the tension or of the depth of the anterior chamber, the type of nephritis was chronic parenchymatous, and histologically there was thrombosis and degeneration of vessels, interstitial hemorrhages and considerable lipoid substances within the tissues of the eye. In 1921, Fleischer (11) reported as spontaneous separation of the choroid a case in which the left eye showed a tension equivalent to 32 millimeters of mercury, and a shallow anterior chamber, with eventual similar involvement of the right eye. While clinical nephritis was not proven, it appears that the study was incomplete, and that a postmortem examination could not be obtained. It is probable that this case may have belonged to the nephritis group, since the clinical picture seems to conform in many respects to the case of Ewetzky.

Schmidt-Rimpler (12) makes the point that paralysis of the sympathetic often gives sustained hypotonus, along with other classical signs of this condition, such as miosis, ptosis, enophthalmos, and unilateral sweating of the face. He suggests that the considerable fluctuation from day to day in the refractive power of the eye might be due to several causes, one of which may be separation of the choroid. On the other hand, there is not a single instance where separation of the choroid has actually been observed in sympathetic paralysis.

Finally, there remains a group of cases usually referred to as spontaneous separations of the choroid, because no cause for the separation can be ascertained. Of this variety, three cases are accredited to Von Graefe (1), six cases to Liebrich (13), one to Von Wecker (14), one to Story (15), and one to Mules (3). In the earlier cases of Von Graefe and Liebrich, no record appears of the state of tension or of the

depth of the anterior chamber, the diagnosis having been made by the identification of choroidal vessels beneath the retinal level upon the mounded separation. Von Wecker gives the reduced tension a differentiating value in diagnosing separation of the choroid, as against tumor. Story and Mules record the tension in their cases as normal, and they make no reference to the depth of the anterior chamber. In our opinion, the possibility of choroidal tumor is not definitely excluded in any one of these cases.

On the purely experimental side, Collins (16) observed in rabbits' eyes, which had been trephined by Kirkpatrick, that there was a granular coagulum in the perichoroidal space. Collins gives credit to Hudson (17) for having first offered the explanation that hypotonus promoted a transudate from the choroidal vessels into the perichoroidal space in those cases in which separation of the choroid was found.

In the case we have to report, the separation of the choroid was spontaneous in the sense that no obvious cause for the condition could be found upon clinical examination of the eyes. So far as we can ascertain, there is in the literature no case to parallel the clinical and histological findings in our case. From the evidence presented below, we believe that separation of the choroid in our case was due to a cause not hitherto recognized.

The patient entered the Massachusetts Eye and Ear Infirmary only once, in 1911, for the purpose of having his right eye enucleated. In order to make the story complete, it was necessary for us to trace his subsequent course up to the time of his death in 1923 through his family, his family physician, his three successive ophthalmologists, and the records of the Peter Bent Brigham Hospital, where he was a patient in 1916. For the sake of convenience, we have assembled the information chronologically.

CASE 1.—SPONTANEOUS SEPARATION OF THE CHOROID.—Until 49 years of age, the patient, J. F. H., male, enjoyed good general and ocular health. Without known cause, he developed at that age a chronic diarrhea with eight to ten stools daily, continuing practically unabated until his death at 66 years of age.

At 54 years of age, following a fall in February 1911,

there was a gradual, painless reduction in the vision of the right eye, progressing to such an extent in four months that the patient was compelled to seek advice. The ophthalmologist who first saw him found "separation of the retina" in the right eye, without recording the exact site of the separation. The vision in the right eye was set down as light perception. The left eye was apparently normal, with an acuity of $\frac{20}{20}$. Reexamination of the right eye six weeks later showed an irritable eye without light perception, tension -3, moderate ciliary congestion, a green and muddy iris, and a deep anterior chamber. Atropine was started at once, and after three days' use the pupil was dilated, leaving pigment flecks on the anterior lens capsule. The anterior chamber was still very deep, and the "retinal" separation was more marked. Three days later the vision of the right eye was recorded as shadows, and there was marked increase in the pain photophobia, lacrimation, and congestion of the right eye. The tension was -2, the cornea cloudy, the anterior chamber was extremely deep, and there were extensive posterior synechia. The pupil measured 4 millimeters, and reacted neither to light nor to mydriatic. The lens was clear. A marked "retinal" separation could be made out nasally and inferiorly, over which areas transillumination seemed defective. The left eye at this time was perfectly normal, the vision being $(\frac{20}{20})$ -75 S $=\frac{20}{20}$. The right eye was enucleated, August 1, 1911, at the Infirmary, six months after sight had begun to fail, and five years after uncontrolled diarrhea had become established.

At the age of 59 years, the patient entered the Peter Bent Brigham Hospital for the relief of his diarrhea, which still troubled him eight to ten times daily. In his history, the features of note were dyspnœa on exertion, edema of the feet and ankles on standing, the raising of frothy sputum in the morning, and the necessity of sleeping on three pillows. He admitted taking three whiskies daily, and considerable Squibbs' Diarrheal Compound (Opium, Camphor, Capsicum, and Chloroform in Alcohol). Clinical examination revealed little noteworthy other than a blood pressure of 180 systolic, and 65 diastolic. Laboratory findings showed a negative blood Wassermann, a slight trace of albumin with rare hyalin casts in the urine, a secondary anæmia with hemoglobin of 80%, a red cell count of 3,750,000, a white cell count of 6500, and the stools showed no ova or parasites.

Examination with the proctoscope showed on the wall of the large bowel about six inches above the anus a pedunculated polyp, which was removed by snare, and found

histologically to be a Papillary Adenoma. Following the operation, and even two years afterwards, there was not the slightest improvement in the patient's diarrhea.

At the age of 62 years, for the first time, trouble began in the left eye in the form of gradually failing vision. Within two months, sight had fallen to light perception temporally only. Each of the two ophthalmologists who examined the eye found separation of the "retina" temporally and below. We were unable to obtain more information about this eye, since the patient died of pneumonia in 1923, at the age of 66 years. There was no autopsy. According to the family, the patient had not consulted an ophthalmologist during the last four years of his life.

PATHOLOGICAL REPORT.

(2078) This report deals with the right eye of the patient, and with minor additions it consists of the notes made by Dr. Verhoeff in 1911. Incidentally, the tissues of the eye were unusually well fixed, so that beautiful details can be obtained in the sections. The eye was fixed for 48 hours in 10% formalin, opened, and then placed for 24 hours in a saturated solution of potassium bichromate.

Gross: The globe, of normal size, and indented because of low intraocular pressure, was opened by equatorial section. The cornea is clear, and measure 12 millimeters in horizontal diameter. The anterior chamber is abnormally deep, actually 4 millimeters, and it is filled with clear fluid. The pupil is 5 millimeters in diameter, and is free from membrane. There is complete posterior synechia, and the iris-lens seems to be pushed sharply backward The filtration angle is free. The choroid and ciliary body are widely separated from the sclera, being attached only at the pectinate ligament, and over an area 4 to 10 millimeters in diameter about the disk. This separation was clearly made out by transillumination of the globe before it was opened. No cyclodialysis can be made out. While there is no definite sub-choroidal coagulum, there is a delicate translucent fibrillated material along the outer surface of the choroid, and at the angles where the choroid leaves the sclera. Nerves and vessels are seen extending across the sub-choroidal space. No similar material is found beneath the retina, which is wrinkled (artefact), and which except for this is closely applied to the choroid. The optic disc is slightly swollen, and the sub-dural space of the nerve sheath is distended. The nerve itself is normal. The vitreous chamber is reduced to about half its normal size, and the vitreous appears normal.

Microscopic: The cornea is normal except for an old facet, where epithelium replaces the gap in Bowman's membrane and the defect in the superficial stroma. The episcleral tissue at the limbus is congested, but is almost free from cellular infiltration. The filtration angle is abnormally wide, due to retraction of the iris. Indicating that the anterior chamber contained dilute serum during life, there is considerable finely granular coagulum over the posterior surface of the cornea, the anterior surface of the iris and lens, thus forming a complete unbroken lining for the anterior chamber. Special staining shows that this coagulum contains in places a small amount of fibrin. The coagulum appears to be more compact toward its exterior, and bordering the corneal endothelium it shows a curious moth-eaten edge. The coagulum is about the same thickness as the cornea itself. There are no cells applied to Descemet's membrane. The ligamentum pectinatum is normal, but the spaces of Fontana contain serous coagulum, as does the canal of Schlemm, which is patent. The iris is firmly fused to the lens capsule, and to those anterior ciliary processes with which it comes in contact. This fusion is not associated with any visible change in the pigment layer, which even in bleached specimens appears normal except for proliferation of capillaries underneath. There is no pupillary membrane. The blood vessels of the iris are congested, and the formation of new capillaries is observed at the base of the iris, and under the pigment epithelium as stated. The stroma is devoid of chronic inflammatory cells, is not atrophic, and it shows small interstitial hemorrhages in the region of the sphincter. Dilute serum and a small amount of fibrin, are present within the stromal spaces, and by special staining the fibrin shows direct continuity with the fibrin of the coagulum on the surface of the iris. The thickness of the iris is, however, little if at all, greater than that of a normal iris. Occasionally along the surface of the iris are seen large bladder-like cells, some with pigment, some without. The ciliary body is edematous, and the dilated spaces even between the muscle bundles are filled with dilute serum. Here and there small collections of round cells are found. The surface bordering on the perichoroidal space is ragged, due to having been pulled away from the sclera. Within the fibrin meshwork there are a few leucocytes, and many cells which are undergoing hydropic degeneration. It is impossible to say with certainty from a study of these degenerating cells whence their origin, whether from chromatophores or endothelial leucocytes. Certainly many of these cells contain pigment, and most of them show one of the many

stages of vacuole formation, from the small scarcely noticeable droplet, to the large single or multiple vacuoles which crowd the nucleus aside. There can be little doubt that many of these cells are degenerated chromatophores.

The ciliary processes are intensely congested, and show numerous small interstitial hemorrhages. There is only slight cellular infiltration with round cells. There is no cyclitic membrane. The lens is normal except for the pigment on its anterior capsule.

The choroid is congested and edematous, its thickness where attached to the sclera being about twice the normal thickness. No coagulum can be demonstrated within the tissue spaces of the choroid. On the surface of the detached choroid, and also on the scleral surface, there is a loose network of fibrin. No serum can be recognized here, although it may probably have been present during life in such dilution that it was not preserved during the preparation of the specimens. Here and there occurs an insignificant infiltration with lymphoid cells. Scattered throughout the choroidal tissue, sometimes enmeshed in fibrin, there are numbers of cells undergoing hydropic degeneration. Here and there focal proliferation of chromatophores occurs within the shreds of choroidal tissue. The pigment epithelium in general is normal, but does occasionally show vacuolization, and at the disc margin colloid degeneration.

The retina is only slightly altered. It is apparently not greatly congested, and there is no serum or fibrin within it anywhere, but there are punctate hemorrhages just behind the ora serrata, and a few minute hemorrhages in the ganglion cell layer of the macula.

The ganglion cells appear normal except for an occasional cell which shows nuclear pyknosis and vacuolization of the cytoplasm. The optic disc is slightly swollen. The subdural space is distended, the dura being separated from the nerve stem about .65 millimeter at a distance of one millimeter behind the sclera. The nerve itself is normal. None of the blood vessels of the eye show degenerative changes.

A striking fact is the total absence of coagulum and fibrin from the vitreous chamber.

Before attempting to explain the separation of the choroid in this case, it seems best to discuss some of the causes of altered intraocular pressure with especial reference to the relation of altered pressure to separation of the choroid.

While our knowledge regarding the production and resorp-

tion of tissue fluids, including the aqueous, is highly imperfect especially from a quantitative standpoint, we know that the aqueous pressure, and hence the intraocular pressure is largely dependent upon

a. the outflow of fluid from the eye,
b. the hydrodynamic pressure in the intraocular capillaries, and
c. the osmotic properties of the blood.

a. Obstruction to outflow causes glaucoma, while too free outflow causes hypotonus. The latter condition is exemplified by the hypotonus resulting from post-operative wound leakage and is a frequent cause of separation of the choroid. This aspect of the subject has recently been discussed by Verhoeff (18) elsewhere, and the ***modus operandi*** for post-operative separation of the choroid explained as follows: "In a typical case of this kind, the anterior chamber reforms the day after operation. Then the wound gives way at some place, the anterior chamber becomes obliterated and remains so for some time. During this time the intraocular pressure remains almost at zero, due to the free escape of aqueous. The pressure within the choroid now being reduced to the same extent, fluid is transuded from its vessels in greater amounts than can be carried off, distends the sub-choroidal space and causes the choroid slightly to compress the vitreous. Within a few days the wound closes, the intraocular pressure rises, and the anterior chamber is reformed. After this happens and after the intraocular pressure becomes normal, the separation of the choroid may persist for a few days or weeks. This proves conclusively that fluid had been squeezed out of the vitreous by the choroid. Gradually the intraocular pressure forces the fluid out of the sub-choroidal space or causes it to be absorbed, thus proving that under normal conditions the intraocular pressure is higher than that in the choroid." Rigidity of the sclera, as suggested by Meller (6) no doubt increases the likelihood of post-operative separation, since this tends to prevent collapse of the sclera and allows the intrachoroidal pressure to become less than in case of a normal sclera.

In rare cases post-operative separation of the choroid persists indefinitely after closure of the wound. To explain such cases, Verhoeff assumes that the intraocular pressure even under ordinary conditions is unusually low in these eyes, so that after closure of the operative wound it does not become high enough to overcome the traction of the zonule and vitreous and push the fluid out of the sub-choroidal space. On this assumption provided the wound has fully closed, the rational procedure, evidently, is to make a scleral puncture and allow the sub-choroidal fluid to escape. Verhoeff (19) has already reported one case in which this procedure was successful, and has subsequently carried it out successfully in the following case:

CASE 2.—POST-OPERATIVE SEPARATION OF THE CHOROID TREATED BY SCLERAL PUNCTURE.—Byron G., aged 76 years, posterior cortical cataracts O.U. September 16, 1921, intracapsular extraction with iridectomy O.S. by Verhoeff method, without loss of vitreous. Patient discharged from hospital September 27. A large separation of the choroid seen October 4 on the nasal side. V.O.S. with $+10.50\ S = +3.00$ cyl. $\times 173° = \frac{20}{100}$. October 11, separation persists, tension very low, V.O.S. with glass $= \frac{20}{60}$. October 18, patient sees semicircular shadow in outer field of left eye. November 3, separation persists. Tension is 10*mm* by Souter's tonometer. V.O.S. with glass $= \frac{20}{40}$. Operation: Scleral puncture with Graefe knife, through conjunctiva 7*mm* behind limbus. Only a small amount of fluid comes out. Incision enlarged, spatula inserted, pressure on sclera with another spatula causes exit of large amount of fluid, and collapse of sclera. Cornea collapses when pressure with spatula is released. Thirty minutes later, cornea wrinkled. Two and one half hours later, globe filled out, cornea and anterior chamber normal, separation of the choroid not visible on brief examination with ophthalmoscope. November 7, a slight separation of choroid can apparently still be seen at the extreme limit of the ophthalmoscopic field, and a small hole can be seen in the choroid. Tension 10*mm*. V.O.S. with glass is $\frac{20}{70}$. No definite separation can be made out, but the hole can be seen. Patient states that vision is always worse in the morning but that he can read newspaper at night. December 16, 1921, V.O.S. with glass is $\frac{20}{40}$. Hole in choroid visible. Tension O.D. = 12*mm*, O.S = 10*mm*.

March 8, 1922, patient writes that vision of the left

eye gradually improved until it was equally good in the morning and evening. October 30, 1922, V.O.S. with $+11.50\ S = +1.25\ cyl. \times 173° = \frac{20}{20}+$. Tension O.U. *12mm.* Vitreous clear. Through the undilated pupil a gray area can be indistinctly seen in the region of the scleral puncture.

Good vision was maintained in the left eye until the death of the patient in October, 1924.

It is interesting that in this case a successful result was obtained in spite of the choroid being punctured. The vitreous evidently obstructed communication between the vitreous chamber and the sub-choroidal space sufficiently to permit the intraocular pressure to keep the choroid in place. Puncture of the choroid could be prevented by employing the belly of a large Knapp needle for making the scleral incision as in Verhoeff's (20) operation of cyclectomy. It is noteworthy that tension in the unoperated eye in this case was only *12mm.*

b. Increase in hydrodynamic pressure of the intraocular capillaries can, without doubt, increase the intraocular pressure, and when associated with obstruction to outflow as in glaucoma, may account for the prodromal attacks and acute exacerbations in the latter condition. This would seem to be an obvious cause for separation of the choroid associated with nephritis, in which the choroidal vessels show marked degenerative changes, leading, presumably, to increased intracapillary pressure. An altered condition of the blood, favoring edema, however, may be a contributory factor here.

Decreased hydrodynamic pressure within the capillaries is obviously an adequate cause for hypotonus, and seems to be the most rational explanation for the hypotonus associated with sympathetic paralysis. On the theory that the sympathetic supplies constrictor fibers to the intraocular arterioles, paralysis of these fibers should cause ocular hypertonus, not hypotonus. The experimental evidence however that the sympathetic supplies such constrictor fibers is conflicting, and is controverted by clinical facts, as one of us has pointed out (18). The observations of Krogh (21) may throw light on this question, as they suggest that the sympathetic contracts the capillaries, in which case paralysis of the sympathetic would dilate the capillaries, thus decreasing the intra-

capillary hydrodynamic pressure, and per se decreasing the intraocular pressure. We regard the fact that sympathetic paralysis is typically associated with hypotonus as almost conclusive proof that the sympathetic has a constrictor effect upon the capillaries of the ciliary processes. It seems possible, although highly improbable, that the hypotonus in our case may have been due to disturbance of the sympathetic system, wherein the uncontrolled diarrhea may also have had its source.

The fact already noted may be emphasized here, that while the hypotonus associated with sympathetic paralysis has been supposed to be a cause for separation of the choroid, no single case of separation of the choroid from this cause has ever been actually observed. The reason for this, we believe, is that in hypotonus due to this cause, the pressure in the vitreous chamber does not fall below the intra-choroidal pressure. That is to say, the secretion of aqueous is not entirely checked, so that owing to the less free outflow from the anterior chamber than from the choroid, the vitreous pressure remains higher than the intra-choroidal pressure.

Contusions of the eye, without rupture, are sometimes followed by hypertonus, more often by hypotonus. As suggested by one of us elsewhere (20), it seems probable that the former is due to increased and the latter to decreased hydrodynamic pressure in some of the intraocular capillaries, either as the result of direct injury to the vessel walls, or to their vasomotor nerves.

c. Alteration in the osmotic pressure of the blood has been shown experimentally to have a marked effect upon the intraocular pressure. Thus, an increase in the salt content of the blood, or the introduction of glucose into the blood will temporarily reduce the intraocular pressure. The hypotonus of diabetic coma is probably due to such a cause. As illustrating an opposite condition, there may be mentioned a striking observation in a single case made by one of us. In the predisposed eye of a diabetic patient, following a sharp reduction in blood sugar, there occurred an attack of acute congestive glaucoma.

It seems to us that factor (c) offers the most satisfactory explanation of the hypotonus and the separation of the choroid

in our Case 1. Although we have no record concerning the tension of the right eye until about five months after the first disturbance of vision, we must assume that the hypotonus preceded the separation of the choroid. For it is not possible to conceive that the latter could have caused the hypotonus, and we know that hypotonus is an adequate cause for separation of the choroid. It would seem that such unusual ocular conditions must have been due to some unusual cause. The only unusual systemic condition manifested by the patient, so far as we know, was persistent severe diarrhea of unknown etiology, and it is therefore logical to conclude that this was either itself the cause, or it was dependent upon the same causes as were the ocular conditions.

It would seem that severe diarrhea of such long duration, at some period at least, would have markedly reduced the watery content of the blood in spite of the allaying of the thirst of the patient by copious water drinking. We assume that a condition finally obtained in which the osmotic pressure of the blood was insufficient to enable the hydrodynamic pressure to force fluid from the capillaries through the ciliary epithelium, while still permitting some fluid to be forced out into the tissues of the iris, ciliary body, and choroid. As a result, the pressure in the vitreous chamber became much lower than that in the anterior chamber and sub-choroidal space, evidence for which may be found in the fact that the iris and lens were pushed far back, and the iris was compressed against the lens and ciliary processes. It may be noted here that the pull of the zonule upon the ciliary body resulting from backward pressure upon the lens, may have been a contributory factor in causing the choroidal separation.

Since the outflow of fluid from the vitreous into the anterior chamber was impossible, it may be asked, how did this fluid escape and allow the vitreous to become compressed? It is evident that the process was very slow, since even after six months the vitreous still maintained half its normal bulk. We may assume therefore that the vitreous fluid was absorbed by the retinal vessels. This absorption would have occurred all the more readily owing to the facts that the vitreous fluid was practically free from albumin, and that the blood was presumably deficient in water.

The intense congestion of the vessels in the uveal tract is to be explained by the hypotonus. Under normal conditions, the intraocular circulation is adjusted to withstand the normal intraocular pressure, and therefore the intraocular vessels are constantly in health under higher surrounding pressure than are the extraocular vessels. When this external pressure is withdrawn upon the establishment of ocular hypotonus, the whole intraocular vascular system would necessarily become distended. This distension, it is to be noted also, would favor the transudation of fluid into the sub-choroidal space and anterior chamber, and explains the occurrence of the slight amount of fibrin in these places. It also explains the proliferation of iris capillaries. The papillœdema in this case is obviously explained by the hypotonus. For the distension of the sub-vaginal space of the optic nerve we can offer no definite explanation.

The most serious objection to this theory is the long interval between the involvements of the first and second eye. It is not difficult to understand that temporary hypotonus might occur in one eye and not in the other, because this could be explained by slight differences in their anatomical structures and vascular conditions, but the real difficulty lines in explaining how one eye could become permanently soft, while the other remained for years unaffected. To explain this we assume that the hypotonus produced conditions that tended to perpetuate themselves. We assume that as in glaucoma, increase in intraocular pressure increases the intraocular capillary pressure, and tends to set up a vicious cycle, so in an opposite way hypotonus tends also to establish a vicious cycle. Under ordinary conditions this vicious cycle would be broken, but in our case where the underlying systemic condition—*i.e.*, chronic diarrhea—persisted, normal intraocular conditions could not be re-established. It is to be noted in this connection, that while the left eye remained unaffected for years, it did ultimately become affected in the same way as the right eye, and that the condition persisted in this eye also. Incidentally, these considerations suggest that in such cases, scleral puncture could be employed with some hope of success.

Another difficulty that presents itself is that while cases

of diarrhea are common, spontaneous separation of the choroid is extremely rare. This difficulty is met by assuming that the exact condition of the blood necessary to check the aqueous secretion and yet permit transudation of fluid into the choroid so seldom occurs that its chance of occurring in diarrhea of relatively short duration would be slight. Even in the present case this condition of the blood apparently occurred only twice during the first thirteen years of persistent severe diarrhea, and the separation of the chloroid in the first eye did not occur until the fifth year of the diarrhea.

It is possible that the complete explanation of the separation of the choroid in our Case 1 is not quite so simple as we have assumed, and that the diminished water content initiated other conditions of the blood which favored the transudation of fluid from the uveal vessels. Present knowledge regarding such possible conditions, however, is so imperfect that it would be unprofitable to attempt to discuss them, and in any case, loss of water from the blood seems to be the essential factor in the explanation.

As regards the clinical diagnosis of separation of the choroid, our experience has been confined to post-operative cases. In these the diagnosis is easily made, provided an ophthalmoscopic examination is possible. When with the ophthalmoscope there is seen a projection at the periphery of the field on one or both sides, or below, presenting a dark smooth surface and a sharp outline, which on transillumination becomes luminous as does a cyst, the diagnosis is certain. Knowledge of previous wound leakage or obliterated anterior chamber is not essential for the diagnosis. According to the anatomical findings in the present and in many cases of soft atrophic eyes, separations of the choroid always involve the anterior part of the uvea, while the choroid tends to remain adherent in the fundus. A localized elevation of the choroid in the fundus should therefore be regarded as certainly not a simple separation of the choroid, and as almost certainly due to a tumor. It would seem, therefore, that in a case of spontaneous separation of the choroid the diagnosis could as readily be made by means of the ophthalmoscope and transilluminator as in a case of post-operative separation.

In conclusion, based on the foregoing considerations, the

following classification of separations of the choroid is offered, and is believed to include all the varieties of clinical importance:

(1) Those associated with increased intraocular pressure and shallow anterior chamber. These are due to local vascular disturbance in the choroid or ciliary body, causing increase in the intra-choroidal pressure, and are exemplified by the cases associated with arteriosclerosis and nephritis.

(2) Those associated with decreased intraocular pressure. These include cases with:

a. Hypotonus and shallow anterior chamber due to wound leakage, as exemplified by post-operative separations.

b. Hypotonus and deep anterior chamber, resulting from ocular vascular disturbance. This variety may be spontaneous as in the cases of sympathetic paralysis, or traumatic, but at present it has only a hypothetical existence, as no case has ever been demonstrated.

c. Hypotonus and deep anterior chamber, due to changes in the osmotic properties of the blood. This variety is believed to be exemplified by the case here reported.

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OPHTHALMOPLEGIA INTERNUCLEARIS.

Report of a Case.

By Dr. EDWIN BLAKESLEE DUNPHY, Boston, Mass.

(From the Department of Ophthalmology, Harvard Medical School, and the Teaching Service, Massachusetts Eye and Ear Infirmary).

CASES of paralysis of lateral associated ocular movements are sufficiently rare to be interesting and worth reporting.

The various types of this paralysis are probably best classified by Duane in Fuchs' test book (1), as follows:

(a) Neither eye when attempting to follow an object that is moving to one side, say the right, can move beyond the middle line. But the left eye can move to the right when converging on an object which is brought in towards the eyes in the median plane (retention of convergence).

(b) Neither eye can follow an object that is carried to the right, provided both eyes are open, but the left eye can follow the object to the right if the right eye is covered. This is really the same case as (a), the movement inwards of the left eye when the right eye is covered being simply a movement of convergence. (Jefferies, Moebius.)

(c) The right eye can follow an object moving to the right but the left cannot. The left eye, however, can move to the right in performing convergence. This condition, which may be called conjugate hemiparalysis, is thus distinguished from paralysis of the left internus in which the left eye can move inwards neither when converging nor when attempting to make a conjugate movement to the right.

(d) Both interni may be paralyzed for conjugate movement (Wilbrand & Saenger.)

(e) Neither eye can follow an object which is carried to the right, and the left eye, moreover, cannot move to the right when attempting to converge (combined paralysis of lateral movement and convergence).

Paralysis of lateral movement is sometimes bilateral; *i.e.*, both eyes fail to follow an object that is carried either to the right or the left, although it may be that both can still converge. The case reported here belongs to type (c) of the above classification.

REPORT OF THE CASE:

Mr. F. W., age 33, clerk by occupation, presented himself at the out-patient department of the Massachusetts Eye & Ear Infirmary, December 1, 1924, complaining of double vision and dizziness. His story was that 5 days previously, after finishing a strenuous game of basket ball, he experienced sudden diplopia, accompanied by great dizziness and nausea. He had severe pain over the left temple which lasted almost continuously up to date of admission. He was seen by his family doctor who ordered him to bed and suggested taking a blood examination, but apparently this was not done.

Past History.—Always in good health; appendectomy, 16 years ago. Gonorrhœa, 14 years ago; denies chancre.

Family History.—Father living and well. Mother died of carcinoma of uterus. One brother living and well. No deaths.

On examination the patient showed the following: The right eye turned out and there was blepharospasm to avoid diplopia but no true ptosis of lid. The eye could not be rotated beyond the mid line nasally when both eyes followed an object carried to the left but possessed perfect converging power along with other eye when object was carried straight in toward nose. When the left eye was covered the right eye could not be rotated to left. In other words, the right internal rectus was apparently paralyzed for lateral movement, but functioned perfectly for convergence. The other movements of the right eye (up, down, and out) were normal.

The excursions of the left eye were entirely normal.

There was slight rotary nystagmus to left when patient attempted to fix with either eye. The vision was $\frac{20}{20}$ with each eye, with slight myopic correction. The pupillary reactions were normal, direct and consensually. Accommodation was unimpaired in either eye. The fundi were normal.

The patient was admitted to the house on the service of Dr. George S. Derby for study.

Blood Wassermann was negative. Spinal fluid Wassermann strongly positive; cells 8; protein 28*mg* per 100*cc:* Gold sol. 0122111000. Complete neurological examination done by Dr. Viets at Nerve Department, Massachusetts General Hospital was negative except for the ocular disturbance.

Barany Test Report.—"Spontaneous nystagmus in 3 planes—all canals readily stimulated and normal eye reactions produced, little stimulus required indicating vestibule is in irritable condition.

"Toxic labyrinthitis from

1. Positive Spinal Wassermann.
2. Bad teeth.
3. Tonsils deep foci."

Dental Examination.—Mouth in poor condition; pyorrhea; X-ray shows lower left molar, lower right 2nd molar, and upper right 3rd molar abscessed. Epulis left superior lateral region.

Nose and Throat Examination.—"Left lower turbinate swollen and red; no pus; right middle turbinate enlarged.

"X-rays of sinuses negative; tonsils ragged."

The patient was given injections of salvarsanized serum by the cistern route at weekly intervals, five treatments being administered.

The abscessed teeth were extracted and the epulis removed by curetting the bone.

Improvement was noted after the first cistern injection. He could use his internal rectus more and more during the next few weeks until finally the condition was cleared up entirely.

Since its first reported observation by Foville in 1859 there have been numerous theories advanced to explain the cause of this rather unusual type of ocular motor disturbance. One popular theory was a lesion in the region of the abducens nucleus as this was supposed to be the center for lateral associated movements.

Lutz (2) refutes this theory and shows that fibers for lateral associated movement cannot arise in this nucleus. Other authors presuppose a supranuclear center in the pons.

More recently Spiller (3) reported a case in which there was a sudden paralysis of both internal recti muscles in associated

lateral movement, with preservation of convergence and preservation of the movements of the external recti muscles. The paralysis accompanied an apparent vascular attack in which there was fullness of the head, numbness of the left side, and indistinctness of speech. The patient died 19 days after the attack, and necropsy showed each abducens nucleus to be intact as well as both oculomotor nuclei. An area of softening had destroyed most of the left posterior longitudinal bundle and had also invaded the right posterior longitudinal bundle along with the left trochlear nucleus.

Spiller's case is the only one of its type so far reported in which histological sections were studied, and definitely places the lesion outside the nuclei and in the posterior longitudinal bundle.

Lutz has suggested the names "Ophthalmoplegia Internuclearis Anterior" and "Ophthalmoplegia Internuclearis Posterior." In the anterior type the rectus internus fails to function in lateral movement but has preserved convergence power, all other muscles being normal. In the posterior type there is only a paralysis of the rectus externus in lateral movement and the diagnosis between a supranuclear or an intranuclear palsy must be made from other concomitant symptoms.

In the case reported here, there was probably a specific endarteritis with vascular occlusion which cleared up rapidly under salvarsan, or possibly a gumma formation in region of the posterior longitudinal bundle without involvement of the nuclei.

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PERCENTAGE VALUE OF VISION AND THE SNELLEN NOTATION.

By Dr. J. A. C. GABRIELS, Albany.

WITH the enactment of statutes whose administration is concerned in the interpretation of the value of visual function, the need of a sound and adequate system of percentage equivalents for the Snellen notations in common use is evident. In the operation of the Workmen's Compensation Law as it applies to ocular injury or disease, this is well exemplified. The endeavor, however, to assign appropriate percentage values to the various degrees of loss in visual acuity has provoked much thought and discussion, with results far from conciliatory.

Given a method based on sound principles, and a satisfactory scale of visual values—applicable alike to industry—should evolve. The following rationale is presented in the belief that it points the way.

According to the Snellen test types, the ability to discern letters subtending an angle of 5 minutes—the component of which is thus distinguished under the angle of 1 minute—constitutes normal acuteness of vision. The distance at which such a letter is seen by the normal eye is 20 feet, and the height of this object at the given distance equals 0.891 centimeter.

Throughout the Snellen visual series a corresponding ratio of increase in angle, distance, and size of object is maintained, —namely, one half of the normal, or 2.5 minutes of angle, 10 feet in distance, 0.4455 centimeter in height of object. Disregarding the tangent, this gives for the serial unit first removed from normal 7.5′ of angle and $\frac{30}{80}$ in footage; for the second 10′ and $\frac{40}{80}$; for the third 12.5′ and $\frac{50}{80}$; and so onwards.

With each such increase in size of angle and in distance there is indicated reciprocally a decrease in the value or efficiency of vision, *i. e.*, the larger the angle or greater the distance, the less the value or acuteness of vision. Therefore from the viewpoint of visual values, Snellen notations progressing from normal represent ***primarily*** losses, and actually express remaining efficiency of vision only ***indirectly***. As will be seen, Sn $\frac{20}{20}$, $\frac{20}{15}$, $\frac{20}{10}$, etc., representing equal and constant increases in distance and size of object or image, do not connote equal and corresponding losses in value of vision. Moreover, $\frac{20}{20}$, $\frac{20}{15}$, etc., denote, in fact, relative values inasmuch as they take into account the standard or norm. For our purpose, then, it is necessary to regard the values indicated by $\frac{20}{20}$, $\frac{20}{15}$, etc., as per se loss values, ***relative*** as considered together with the norm and ***absolute*** when determined with reference to a fixed point of blindness. Once having established the relative value of each successive 2.5 minutes of angle increase in size of object, its reciprocal or the decrease relative value of vision is simultaneously deduced.

What, now, is the value of the first 2.5′ deviation from the standard 5′ angle, *i.e.*, from the 5′ to the 7.5′ angle ($\frac{20}{20}$ to $\frac{20}{15}$)? Given the 7.5′ angle, obviously this 2.5′ must have a value equal to one third, and represents therefore a one third increase in angle or distance value, and reciprocally and equivalently a one third decrease in the corresponding value of vision. The 2.5′ angle increase indicated in the formula $\frac{20}{20}$ (7.5′) expresses, then, a reduction or loss in relative value of vision of one third or, decimally, 0.333 plus. In the next advance, with a given angle of 10′, 2.5′ has here an increase-angle or decrease-vision value of only one fourth, and expressed in the symbol $\frac{20}{15}$ represents a relative loss-value in vision of one fourth or, decimally, 0.25. Likewise, in the succeeding angle increase to 12.5′ ($\frac{20}{30}$), the reduction or loss-value in vision equals one fifth or 0.20. Continuing, the loss for $\frac{20}{25}$ is one sixth or 0.166 plus, for $\frac{20}{30}$—one seventh or 0.142 plus, for $\frac{20}{35}$—one eighth or 0.125, for $\frac{20}{40}$—one ninth or 0.111 plus, for $\frac{20}{100}$—0.10;—through successive steps of the Snellen structure.

It may be readily observed that the visual scale shows a steady diminution in relative value,—that the declining values are never the same or equal, are uniformly less and less, and

thereby serve admirably to mark the manner of visual lapse, as well as pointing out the comparative importance of one or the other phase in vision as we proceed away from the norm.

Furthermore, careful analysis of the descending values thus brought forth reveals at once the principle underlying the mechanism of visual loss, viz., that the decrease in value of vision is in inverse proportion to the increase in size of object. To wit:—At $\frac{30}{100}$ (25′) the object is twice the size of the object at $\frac{30}{60}$ (12.5′), the relative value of vision at $\frac{30}{100}$ is 0.10 and at $\frac{30}{60}$ equals 0.20,—therefore the former is twice the size and one half the value of the latter, *i.e., inversely proportional.* Similarly, at $\frac{30}{120}$ (10′) the relative value is 0.25 and at $\frac{30}{200}$ (50′) equals 0.05,—the latter is five times the size and one fifth the value. The same is true for other such ranges.

It is recognized that so far we have had to do entirely with values relative in nature and content. How, now, are we to ascertain and apply absolute values to the various degrees of visual loss designated by the Snellen notation? Properly to measure such values, it is necessary—*first*, to take into consideration not only the standard or norm but also a standard or point of blindness, —*secondly*, to know the sum-total of relative loss-values in visual acuity contained within the range of these limits,—and *finally*, to determine the relation of this total with the aggregate value of any given range in the visual scale. The percentage losses arising out of this conception should be a true and accurate expression of the reduction in value for any particular part of vision.

To fulfill the prescribed conditions, a lowermost limit of central acuity of vision must be assumed. What shall the accepted limit be? Since we are concerned only with such degree of vision as possesses the property of being measured, it must be greater than absolute blindness, *i.e.*, loss of light perception. And, inasmuch as points more or less proximal to this might be chosen on the basis of being measurable quantities, they would be lacking those attributes essential to the proper concept of direct acuity of vision. Moreover, that point should be selected which best expresses the lower limit for all *practical* purposes. Here, as might be expected, a difference of opinion prevails. However, of points variously suggested, Sn $\frac{30}{850}$, $\frac{10}{200}$ ($\frac{30}{400}$), and $\frac{10}{250}$ ($\frac{30}{800}$) may conceivably

be regarded as sufficiently inclusive. Objection may justly be taken to more remote points for the reasons briefly outlined.

Let us employ, for example, Sn $\frac{20}{350}$ as our lower limit. We find that with this range the sum-total of relative losses is, decimally, 2.646 plus. The comparison of this total with any partial loss (the whole and the part) gives us the absolute percentage loss sought. Specifically, the relative value for $\frac{20}{60}$ is 0.333 plus; 0.333 plus divided by 2.646 plus equals 12.6%. The absolute percentage loss for $\frac{20}{60}$ is, therefore, 12.6%. The percentage loss for $\frac{20}{40}$ is found by comparing the cumulative relative value of this range with the sum-total of relative values, *i.e.*, 0.333 plus added to 0.25 and divided by 2.646 plus, or 22.0%. The absolute loss for $\frac{20}{80}$ equals 29.6%, *i.e.*, the aggregate relative value of 0.333 plus added to 0.25 added to 0.20, or 0.783 plus divided by 2.646 plus. In the same manner, the percentage loss is computed for $\frac{20}{60}$, $\frac{20}{70}$, et cetera.

Study of a complete tabulation of absolute percentage loss in visual acuity also clearly discloses how the value of central vision falls,—how, for instance, in the early phase the loss sustained is attended by a sharp rise, whereas approaching more and more towards accepted blindness a definite leveling influence is appreciated, and the comparative differences in value gradually are reduced to a minimum.

Viewed on industrial grounds, the problem resolves itself in the main to the selection of an adequate lower limit and percentage ratings for the early losses in keeping. As establishing a point of economic blindness, the weight of experience and reliable opinion has at different times looked with favor on points reaching all the way from Sn $\frac{20}{100}$—more generally Sn $\frac{20}{200}$—to such removed limits, ordinarily considered in conjunction with practical central vision. Notwithstanding, scientific evaluation and routine observation should, it seems, combine to render the selection of a lower limit for practical vision as well as an equitable point of industrial blindness not an insuperable difficulty.

It will be noted, in referring to the abridged tables of visual value following, that—on the basis of the given variously interpreted lower limits for practical central acuity of vision—percentage ratings for the respective early losses show only slight variation. In this connection, the fact must not be lost

sight of that normal acuity of vision makes for *easy, comfortably sustained, maximum efficiency* and, in the very nature of things, this normal optimal working efficiency is definitely and permanently interfered with by the first losses:—otherwise, one might fairly contend that such slighter degrees of defective vision be altogether disregarded, as not encroaching upon earning ability so-called. The percentage ratings for the first losses in central acuity of vision stand, therefore, as *founded in fact.*

ABIDGED TABULATION OF PERCENTAGE LOSS OF VISION CORRESPONDING TO SNELLEN NOTATION

Lower Limit—Sn $\frac{20}{800}$	– Sn $\frac{20}{400}$	– Sn $\frac{20}{500}$
$\frac{20}{80}$ – 12.6%	$\frac{20}{80}$ – 12.0%	$\frac{20}{80}$ – 11.1%
$\frac{20}{40}$ – 22.0%	$\frac{20}{40}$ – 21.0%	$\frac{20}{40}$ – 19.5%
$\frac{20}{60}$ – 29.6%	$\frac{20}{60}$ – 28.2%	$\frac{20}{60}$ – 26.1%
$\frac{20}{100}$ – 54.0%	$\frac{20}{100}$ – 51.4%	$\frac{20}{100}$ – 47.6%
$\frac{20}{200}$ – 79.3%	$\frac{20}{200}$ – 75.5%	$\frac{20}{200}$ – 70.0%
$\frac{20}{300}$ – 94.3%	$\frac{20}{300}$ – 89.8%	$\frac{20}{300}$ – 83.1%
$\frac{20}{800}$ – 100.0%	$\frac{20}{400}$ – 100.0%	$\frac{20}{400}$ – 92.6%
		$\frac{20}{500}$ – 100.0

SUMMARY.

1. Snellen notations are, fundamentally, relative values.

2. Snellen notations primarily represent losses in visual value or efficiency. By deduction, the remaining visual value or efficiency expressed is concurrently set forth.

3. Snellen notations in numerically uniform ratio do not denote corresponding visual values.

4. The relative values are expressions of the relationship of any constituent part with an incomplete whole (*i.e., part to part*).

5. The absolute percentage loss values express the relationship of any given partial loss with the whole or total loss (*i.e., the whole and the part*).

6. The absolute value corresponding to any Snellen notation is determined on the basis of measurable practical visual acuity, and varies according as the lower limit for this is conceived.

7. The relative value for any given Snellen expression, appertaining as it does to the norm, is constant and unchangeable.

8. From the industrial viewpoint, both horns of an annoying dilemma may be taken in hand—that is, a satisfactory point of blindness and suitable ratings for the first losses.

CONVENTION OF ENGLISH-SPEAKING OPHTHALMOLOGICAL SOCIETIES.

By Mr. H. DICKINSON, London.

THIS Convention, which was favored by large contingents from the United States, Canada, and the Netherlands, as well as several from the more distant Antipodes, was held at University College (University of London) on July 14th, 15th, 16th and 17th, under the presidency of E. Treacher Collins.

The work of the Convention was divided into two simultaneous sessions for the hearing and discussion of papers; while for two special events combined meetings took place. These events were a symposium on "The Evolution of Binocular Vision," and a debate on "The Microscopy of the Living Eye." The opening ceremony, the presentation of the Nettleship Prize, and the President's address took place in the Memorial Hall of the College.

The Minister of Health's Welcome.

The Rt. Hon. Neville Chamberlain, the Minister of Health, extended, on behalf of his Majesty's Government, a warm welcome to the delegates, especially those who had come from overseas. He said it had not been found possible yet to resume the international gatherings, owing to the backwash of the war, a circumstance he was sure was generally regretted; but those attending the Convention would find it conducted in a tongue that all could follow, and it had brought together peoples from the British Dominions and the United States, who were already united by such friendly ties. He referred with satisfaction to the great advances

which had been made in ophthalmology during the last seventy years, and the efforts being made in this country, in the 482 ophthalmic clinics, to deal with and prevent eye disease, especially in the young, to whom sight was of such vast importance. Under the presidency of Mr. Treacher Collins, whose name was so well known among ophthalmologists the world over, the Convention was bound to be a great success, and he believed members when they departed would be armed with a new vigour and confidence for the warfare on behalf of the most precious of the special senses.

His Excellency the AMERICAN AMBASSADOR expressed, on behalf of his American brethren, thanks for the welcome which had been extended to them, and to the British committee for the arrangements made for their instruction and comfort. He then, in playful mood, deplored the fact that so many of the inhabitants of the States were being left without eye treatment while the ophthalmologists were disporting themselves in England, and uttered a word of warning as to the possible results of the feasting and entertainment provided.

Representatives of the various bodies composing the Convention were then separately presented to Mr. Chamberlain, who warmly greeted them.

Presentation of the Nettleship Prize.

The PRESIDENT then presented to Prof. S. E. Whitnall, of Montreal, the Nettleship Prize, which is awarded triennially for the encouragement of scientific ophthalmic work, for his work on the anatomy of the orbit and accessory organs of vision. The recipient made a graceful acknowledgment.

Critchett Memorial Presidential Badge.

On behalf of a number of intimate friends and colleagues of the late Sir Anderson Critchett, Mr. W. T. Holmes Spicer invested the President with a handsome presidential badge, worked in gold and enamel, bearing the portraits of Sir Anderson and his father, George, and a Latin motto. Mr. Spicer recalled a number of personal characteristics and inci-

dents concerning both, who, together, had practised ophthalmology for nearly a hundred years.

The more recreational items embraced a banquet in the historic City Guildhall, a reception at the Royal College of Surgeons, a garden party (given by Sir William and Lady Lister), a reception at the Wellcome Historical Museum at the Royal Institution of Great Britain, and visits to a number of ophthalmic hospitals in and near the Metropolis. In addition a number of members visited Oxford and Cambridge Universities.

The Bowman Lecture was delivered by Sir John Parsons, who delivered a learned treatise on "The Foundations of Vision." In connection with the Convention there was a remarkably complete museum.

Appended is a short account of the symposium, the general discussion, and some points from the numerous papers read in both sessions.

The President's Address on the Elimination of Eye Disease.

Mr. TREACHER COLLINS, in his presidential address, said his purpose was to give some historical facts which, aided by imagination, might enable peeps to be obtained of some of the wonders which might some day be brought to pass. Work which had recently been done made it possible now to contemplate the elimination of some eye diseases from the human organism.

In leprosy the eyes were frequently affected, and though active measures had rendered it practically non-existent in England, the Empire was said to contain 300,000 cases of the disease.

Dealing with small-pox, he said that about half the cases of indigent blindness applying for relief in this country in the last century lost their sight through small-pox, but in 1922 a careful enquiry revealed only six cases due to the disease in the various institutions for the blind. Much of this improvement was due to the greater care of the eyes in all pyrexial diseases, and not only in cases of small-pox.

Ophthalmia was carried by an organism which did not

produce spores, and as drying killed the organism, infection could only occur while the eyes had a moist discharge. Trachoma, the worst form of ophthalmia, had not had any one micro-organism identified with it, but it was possible that here also the virus was killed by drying it, and measures based on this idea were largely responsible for the practical elimination of the disease from the British Army and the Poor-law schools. At an inspection of Poor-law schools conducted by Edward Nettleship in London in 1874, 42% were found to have granular lids or trachoma, but in 1896 Sydney Stephenson could find only 4.91% of such sufferers.

Syphilis was held to be answerable for from 10% to 15% of the eye disease of the country, but Mr. Collins considered that in the struggle against the spirochaeta now being waged man was bound eventually to win, especially after the "conspiracy of silence" concerning the disease was broken by the Report of the Royal Commission on the subject. So serious were the consequences of syphilis that the President considered that when any person communicated it to another wilfully he should be incarcerated and treatment carried out until he was no longer infectious.

The influence of metastatic septic infection as a cause of iritis was now largely recognized, thanks to the convincing labors of William Lang, who found that in 37% of his cases of iritis, pyorrhœa alveolaris was the only ascertainable malady.

With regard to tuberculous eye disease, Mr. Collins said he was not able to establish by figures that these were diminishing of late, but both in America and here the death-rate from respiratory tuberculosis showed a gradual drop. He was able to speak, from a long experience of the Swanley Ophthalmia Schools, of the great benefit sufferers from tuberculous eye disease derived from an open-air life, with good food and plenty of recreation.

Phlyctenular ophthalmia and its elimination was a social problem, closely associated with bad housing and congestion of the population, also with dietary errors, especially the absence of the fat-soluble A vitamin.

He had something very definite to say on the prevalence of myopia, declaring that it was largely the outcome of our pres-

ent system of education. It was unnatural to put the young growing animal to pore over books for several hours a day, often in artificial light, and he thought more use should be made by the education authorities of the cinema, the lantern, object-lessons, and oral teaching. Lessons in geography, for instance, could take the form which would appeal to the young mind as a veritable voyage of discovery.

He made reference, in conclusion, to the work of the Council of British Ophthalmologists, the similar body in the United States, and the ophthalmic work of the League of Nations and the Red Cross Societies League.

Symposium on the Evolution of Binocular Vision.

At the symposium, largely attended, the chair was occupied by the President, and the first speaker was PROFESSOR ELLIOT SMITH, F.R.S., who said that anatomists could throw some light on certain aspects of this problem, or could at least define issues which others could more fully interpret. The fundamental fact which brought about the evolution of the primates was the cultivation of vision, and the increased reliance on it for guidance. It led to the performance of a greater range of movements, some of them skilled, the development of which in man placed him in his position of supremacy. Fortunately, for the sake of this enquiry, in the case of the primates there were a series of creatures which had survived and escaped the devastating effect of extreme specialization. The primates came into existence in North America, near Wyoming, before the beginning of the Tertiary period, where they separated from the group of insectivorous animals. The retinæ, cerebral cortex and oculo-motor nuclei of these primates had been closely studied by Mr. Le Gros Clark in Borneo and other places. Shortly before the beginning of the Tertiary period the lemurs came into existence, and in one class of them smell began to be replaced by vision as a guiding sense. The tarseus was a very important animal to study in this connection, as it had survived from the beginning of the Tertiary period with practically no change. The speaker was able, some years ago, without histological technique, to map out the distribution of the cor-

tical areas in a series of animals, and to show that, commencing with the jumping shrew, there was a gradual replacement of smell by vision, and since that time Mr. Le Gros Clark, by histological methods, had confirmed the work. In the marmoset this supplanting of smell by sight was very marked in comparison with tarseus. The chameleon illustrated in a striking way the pre-binocular vision stage, as this animal could look in a forward direction with one eye, and, with the other, in a backward direction. A true macula lutea was found only in the apes and man.

Sir Frederick Mott, F.R.S., in his contribution retraversed the ground he covered in his Bowman Lecture of nearly twenty years ago on the evolution of the cerebral cortex and binocular vision in mammals, contending that later researches had supported his thesis. Some slides he showed proved that sometimes the lunate sulcus, seen in all anthropoids, was occasionally met with in the human. With the rise of creatures in the scale, the peristriate area of the occipital lobe was seen to be increased, and the frontal region of the brain underwent increased development. Professor Watson, of Baltimore, found that a baby could fix a light with its eye soon after birth, but the hand movements in association with this fixation were not acquired until the 130th day of life. In the full-term fœtus there was a myelination of the optic radiations, and this was considered to be an agent in the fixing of the light. Sir Frederick contended that all our visual experiences were associated with tactile perceptual ones, and he showed a series of slides which illustrated work which Professor Sherrington and he had done together.

Prof. S. E. Whitnall (Montreal) said this problem had interested him chiefly from the point of view of the movements of eye muscles during the gradual transition of the eyes, in succeeding species, from the side to the front of the head. Lemurs, and all animals below them in the evolutionary scale, moved their heads to see objects, instead of moving their eyes, and the extent to which human beings moved their heads was not appreciated until they suffered from a stiff neck. If in order to increase the visual field of an animal the eye was not moved, the orbit could be moved; while herbivora had an elongated pupil to increase the avenue of vision. He

dealt in detail, by means of diagrams, with the changes in the visual in relation to the median line of the head, and the alterations successively seen in pairs of optical muscles. He said that many animals converged while feeding, but they did not possess stereoscopic vision.

Sir Arthur Keith, F.R.S. (of the Royal College of Surgeons) in view of what had been said by previous speakers, dealt almost solely with the main modifications in the skull and orbit in the process of reaching towards the acquisition of bifoveate vision—a term he preferred to binocular vision. Man probably took his human form in the Miocene period, but his ocular outfit had been acquired long before that time. He said that one of the greatest services rendered to anatomy was Prof. Elliot Smith's clear proof that the evolution of bifoveate vision was the result of sight gradually replacing smell as the dominant guiding sense. Sir Arthur thought this development was closely associated with the mode of climbing, for all bifoveate primates were jumpers, and they must be able to judge the distance of the branch they wished to reach. Twenty-four years ago, Lindsay Johnson showed that monkeys, anthropoid apes and man had the same form of eye and retina and the same ocular muscle outfit. In ordinary monkeys head and eyes moved together, and the power of lateral movement of the eyes was small in them. In the great anthropoid ape conjugate movements were free and frequent, but convergence could not long be sustained. Man's superiority lay not so much in the kind of eye with which he was furnished but in the quality of brain behind the eyes. Still we lacked knowledge of the actual machinery which transformed the visual world as seen by the marmoset into that as seen by man.

On the proposition of Sir George Berry, M.P., seconded by Mr. J. B. Lawford, the speakers in the symposium were very cordially thanked.

Discussion on "The Microscopy of the Living Eye."

On Friday morning the Sessions combined for a discussion on this subject, separate speakers dealing with different portions of the eye.

W. GORDON BYERS (Montreal) spoke about the cornea. He said that though the direct method of examination gave aids of great value, limits to its effectiveness were imposed by great difficulties of illumination. The value of the new method was chiefly in disclosing changes collectively, giving a meaning which could not be attached to isolated findings. Pathological changes were now to be seen in the cornea with great distinctness, because of the variety of illumination now possible. Still, the slit-lamp revelations were probably less important in regard to the cornea than in respect to some other parts of the eye.

ARTHUR BEDELL (Albany, New York), who dealt with the vitreous, said the slit-lamp offered a method for a more careful study of vitreous detail than had previously been possible. In disease, the changed framework might consist in the massing of fibres and the liquefaction and absorption of structures, also in pigmentation. The slit-lamp had made possible an exact prognosis in cases of penetrating injury, as it revealed the presence or absence of tension fibres, which might later cause a detachment. He showed a wealth of slides and much detailed information.

T. HARRISON BUTLER (Birmingham) spoke of the lens. He said the retro-lental space was a cleft filled with aqueous, and in cases of degeneration of the vitreous this disappeared. He showed drawings illustrating detachment in lens dislocation, in glass-blowers' cataract, and in an instance of shrunken lens. He said the lens sutures were known to constitute the sites of various kinds of cataract. The lens shagreen, he considered, must be due to the intimate structure of the capsule itself. He showed that an abnormal growth of the lens zones caused central cataracts and varied forms of zonular or lamellar cataract.

BASIL GRAVES went into the optical properties of the new apparatus, and gave a number of illustrations by means of the epidiascope. He said it was desirable for the observer to train himself to observe the depth of the anterior chamber. The slit-lamp should be so constructed that the vertical width of the beam at its focal region could be made to cover the full diameter of the cornea and anterior chamber. The study of the aqueous fluid resolved itself into two factors:

(a) observation of the outstanding beam, (b) observation of discrete particles.

IDA C. MANN dealt with the **embryology of the lens,** and the correlation of it with **slit-lamp appearances.** At the first stage she showed there could be seen the beginnings of the free cells whose persistence was said by Vogt to be the cause of anterior axial embryonic cataract, which was named after that authority. But examination of this and succeeding stages showed this to be unlikely as the opacities described in the adult lens were in the vicinity of the anterior Y-suture, while the potential cavity of the lens vesicle was immediately under the anterior lens epithelium. She described the anterior vascular capsule of the lens when seen with the slit-lamp.

T. HARRISON BUTLER gave elaborate pictorial representations of some **changes** observed with the **slit-lamp** in **eyes** which had either received an **injury** or had been **operated upon.** After a blow on the eye the lens was found to be displaced backwards, as it was also after penetration by a steel fragment which was removed by a giant magnet. Glaucoma occurred some months after the operation, in each case, and this glaucoma was cured by paracentesis. Radial ruptures of the iris were not discovered until the eye was examined by focal light. In three of his cases, examination with the slit-lamp gave timely warning of an approaching sympathetic ophthalmitis, and prompt measures taken at this stage resulted in a complete recovery.

R. C. DAVENPORT (London) reported on **Recovery of Visual Field in Pituitary Tumors without Operation** in two cases of pituitary tumor with primary optic atrophy and temporal hemianopia. The X-ray showed extensive destruction of bone in the pituitary region. The cases were treated with X-rays with improvement of visual fields but no change in the visual acuity.

THOMSON HENDERSON (Nottingham) spoke on **The Postural Activity and Evolution of the Ciliary Muscle in Mammalia; A Study of the Mechanism of Accommodation,** and drew attention to the fundamental physiological feature that the ciliary muscle's tonic muscular action and not choroidal elasticity holds the zonule taut and acts as a counterweight to the lens.

The ciliary muscle gives a perfect illustration of what Sherrington describes as "postural activity," which as its name implies is the tonic activity of the muscles, whether striated or smooth, whereby they maintain the postures or configurations of the body. An appreciation of this fact that the zonule is kept taut by postural activity of the ciliary muscle, makes it possible to see any purpose in the diversity of the anatomical details at the angle of the anterior chamber in mammals.

A. F. MacCallan (London), in **The Treatment of Trichiasis and Entropion Resulting from Trachoma,** states that there are three types of operative procedure only required to enable a surgeon to deal successfully with all varieties of these conditions. The operations are:

The mucous membrane graft operation of Van Millingen; the excision of a wedge-shaped strip from the tarsus of Streatfield; and the extirpation of the tarsus operation of Saunders.

Alan C. Woods (Baltimore, Md.) in a paper on **Sympathetic Ophthalmia; The Use of Uveal Pigment in Diagnosis and Treatment** stated that the diagnostic tests are a complement-fixation reaction in the blood serum against an antigen of uveal pigment, and an intradermal test to determine specific hypersensitivity to uveal pigment. The serums of 71 patients with traumatic cyclitis have been examined in the complement-fixation reaction; 34 of these have given positive results, and 37 negative.

The intradermal tests for specific hypersensitivity has been used 30 times giving positive results in 16 instances and negative results in 14.

The use of oveal pigment in the treatment of sympathetic ophthalmia was discussed. The technical methods of preparing a pigment were given and the general methods of administration outlined.

J. Herbert Waite, George S. Derby and E. B. Kirk (Boston, Mass.) reported on **The Light Sense in Early Glaucoma.** The authors feel the need of more delicate tests for the early diagnosis of glaucoma and along these lines have been experimenting with the light sense. The paper discusses the difficulties of the examination of the light sense, with a criticism of the previous studies. A special apparatus

is described which gives a constant intensity and which can be exactly varied and recorded over a range of 300,000 to 1. Tests for the light minimum and for the light difference were then carried out. In established glaucoma they find that the dark adaptation is markedly retarded and the threshold is considerably higher than in the normal eye. There was no difference in the light difference in glaucomatous eyes.

LESLIE PATON and IDA C. MANN (London) illustrated by lantern slides **The Development of the Oculo-motor Nuclei and its Bearing on the Argyll-Robertson Pupil Reaction.**

J. VAN DER HOEVE (Leyden, Holland) spoke on **glioma retinæ** and showed photographs of glioma metastases in different parts of the head after binocular enucleation. In another case, in a child aged three the radiograph showed the optic canal on the affected side to be twice the size of the other and normal size. He thinks that the enlargement of the optic foramen is important in prognosis. He concludes that radiography of the optic foramen should be more frequently carried out, and though radiotherapy may be of value in glioma it has not produced many definite cures.

J. BURDON-COOPER (Bath), **The Behavior of the Crystalline Lens System in Artificial Cycloplegia and its Influence on the Refraction of the Eye,** found in the greater number of refraction cases tested both with atropin and without that the total hypermetropia under atropin was less than the manifest hypermetropia without when the eyes were tested binocularly. He attributed this to a forward displacement of the lens system as a whole.

H. MAXWELL LANGDON (Philadelphia) in **The Blood Chemistry of Patients with Primary Cataracts** gave the results of a study of blood sugar and sugar tolerance, urea nitrogen and creatinin, which were mostly negative except that several cases of potential diabetes which showed no other signs were discovered.

A. H. SINCLAIR (Edinburgh) dealt with the subject of **intracapsular extraction of cataract,** and described the type of operation he had recently been carrying out with a new pattern lid-retractor, double cross-action forceps, and a new lens expressor and wound guard. When using the latter there had been no loss of vitreous in any of the twenty-one cases in

which it had been employed. He gave a very interesting cinematograph demonstration of the actual operation.

A. E. DAVIS (New York) contributed a paper on the **lens antigen treatment of cataract,** in which he contended that by the use of this antigen alone practically every case of cataract in its incipiency could be arrested, but mature senile cataracts he preferred to take away by surgery. The longest time during which he had had these cases under observation was four years, and the average loss of vision did not exceed 12%.

P. CHALMERS JAMESON, of Brooklyn, placed before the Convention the principles of **muscle recession** with scleral suturing, as applied for the correction of squint. He emphasized the importance of the site of the conjunctival incision, namely, at or about the curve of the semilunar fold. The muscle and the capsular flap should not be injured on the surface which was adjacent to the caruncle, and the scleral site should be carefully cleared of debris. He discussed the question at some length.

PERCY SARGENT and E. C. CARMICHAEL (London) dealt with the **treatment of pituitary tumors,** as they considered that the time had arrived when ophthalmologists should seriously consider what was to be gained by operation. They set out five classes of tumor, remarking that the adenomata were the most frequently occurring and the most amenable to treatment. Moreover their diagnosis could usually be arrived at early. It usually manifested itself by visual disturbances, at first by an equal affection of both eyes, the visual loss involving first the upper and then the lower temporal quadrant. The authors discussed in detail the risks from attempts at removal by operation.

GORDON HOLMES and LESLIE PATON showed the ophthalmoscopic changes which occurred in **cerebro-macular degeneration of the Batten type.** In most of the cases the retinal changes were fairly well marked when the patients came under observation. In very early cases there was seen a small whitish area at the macula, and this was surrounded by a reddened ring. At a later stage, wavy radiating white streaks could be seen on the retina, and later still there was dotting of the macula. In the third stage the evidences of

retinal atrophy spread out to the periphery. Clinically the cases were closest allied to the group of amaurotic family idiocy.

ERNEST CLARKE (London) passed in review the various suggested **causal factors in myopia,** and said his view was in line with that of Sir Arthur Keith, that myopia was an inherited disorder which influenced the sclerotic at its early period. He threw out the suggestion that some extreme cases of myopia were a manifestation of a disease of the coats of the eye, myopia being a sequel and not a cause.

PARK LEWIS (Buffalo) discussed the scope and possibilities of **preventive measures in ophthalmology**, stressing the point that such work must be co-operative. The measures should include the prenatal treatment of interstitial keratitis, and early and thorough treatment of syphilitic pregnant women.

S. LEWIS ZIEGLER (Philadelphia) gave the **History of Discission of the Lens** with the technique of complete discission by the V-shaped method.

F. A. DAVIS (Madison, Wisconsin) discussed **Hereditary Eye Defects in Rabbits, Experimentally Induced.** The lesions were produced by an injection of lens sensitized sera and pulped lens into pregnant rabbits. The study embraced 152 eyes of which 75 were defective. The defect consisted in coloboma of iris, ciliary body, choroid, retina, and at times, optic disc, accompanied by grades of microphthalmos with or without cystic staphyloma. The theories as to the cause of the persistence and overgrowth of mesoderm in the cleft are discussed.

L. WEBSTER FOX (Philadelphia) presented a historical review of conical cornea and its surgical treatment, and described an improved operation for the condition. The operation consists in the excision of a semilunar strip of cornea, an iridectomy, and suture of the corneal wound with silk. He said that occasionally after the operation glaucomatous symptoms arose, but only very rarely did a shred of iris become entangled in the wound. In one case there occurred opacity of the lens, but this was remedied by needling. His experience of the new operation in forty cases had been very encouraging.

ARNOLD KNAPP (New York) reported on the **late results of**

intracapsular cataract operation. Of 200 successive cases operated on between 1910 and 1919 according to the method of preliminary subluxation with the blunt capsule forceps described in the ARCHIVES OF OPHTHALMOLOGY in 1915 and 1921, 85 cases were re-examined with the following results: If the primary result was good, excellent vision was retained, without any degeneration of the vitreous, remarkable freedom from glaucoma, from retinal detachment and from the sequelæ of irido-cyclitis, except for sclerosis of the cornea in two cases.

H. HERBERT (Hove) developed a new glaucoma theory, in which he propounded the question as to whether many chronic glaucomas are not due mainly or entirely to a defective inward pull on the pectinate ligament. There is normally a constant double pull on the ligament muscular and elastic. In many eyes the well developed ciliary muscle extends so far forward and is so strongly connected with the pectinate ligament that the inner two-thirds of the muscle must, in its contraction, exert a definite inward traction on the ligament, separating its lamellæ, and thus facilitating the flow of aqueous through the ligament. But in some eyes the combined pull is scarcely at all inwards; it is almost entirely meridional. This is due to two defects: (1) The muscle does not extend sufficiently far forwards; and (2) the inner portions of the muscle, often relatively ill-developed, are but feebly connected with the pectinate ligament. These defects constitute an anatomical predisposition to glaucoma, which becomes effective when the traction, both muscular and elastic, becomes enfeebled by senile degenerative changes. Inward muscular pull on the ligament appears to supply the only real satisfactory explanation of the reduction by eserin of the tension of the normal eye.

J. HERBERT FISHER (London) discussed **Intra-ocular Lymph Paths,** the channels by which the intra-ocular nutritional fluids leave the eye, more particularly the nutrition of the vitreous, the diffusion of fluid through it, and the channels by which it is removed. The author stresses the importance of the posterior channel of lymph exit, and less on the circumlental space and the angle of the anterior chamber. Glaucoma following thrombosis of the central vein of the retina is also considered.

R. Foster Moore (London). **A Case of Unruptured Intracranial Aneurism which had Caused Rupture of the Optic Nerve and which was Diagnosed During Life.** A man of 35 suddenly developed a severe frontal headache and lost the sight of the right eye 24 hours later. A tentative diagnosis of aneurism was made bringing the loss of sight into relation with a pressure from the aneurism, and at the autopsy, seven weeks later, a mycotic aneurism, about the size of a small cherry, was found involving the intracranial portion of the ophthalmic artery, which had caused complete rupture of the right optic nerve; there was no leakage from the aneurism. All the signs of infective endocarditis were present.

E. Marx spoke on **Simultaneous Registration of Intra-Ocular and Intra-Spinal Pressure,** giving a short account of experiments on simultaneous registration of intraocular and intraspinal pressure under several conditions. Attention is drawn to the similarity of the fluids of both, and also the relation which exist between them. The alterations in pressure in the eye and cerebro-spinal fluid usually vary in the same direction after the injection of sodium chloride; and in opposite direction after the injection of glucose. Pressure changes in the cerebro-spinal fluid are usually more intense than in the eye.

M. S. Mayou (London), in **Siderosis**, attempts to explain the staining of the tissues of the eye as the result of a piece of iron remaining embedded in it, and then describes the changes, particularly in the pigment epithelium, which result. The iron, the author believes to be in a colloidal state, hence it causes peculiar changes in the epithelium of the eye structures. The iron in siderosis is in a colloidal form, which accounts for its slow diffusion.

Malcolm Hepburn (London) read a paper on the **teaching of ophthalmology,** contending that the teaching of the subject should vary in completeness with the different classes of students, which classes he placed into three categories: (1) undergraduates, (2) general practitioner post-graduates, (3) post-graduates intending to specialize in the subject. He criticized a number of practices now carried out, and formulated a scheme making for greater efficiency.

E. L. Jones, of Cumberland, U. S. A., read a note on the

advantage of immediately producing a maximal physiological **lymphatic reaction in eyes** which had been dangerously wounded. He contended that lymph was Nature's chief agent for combating infection and for repairing injured tissues. By injecting under the conjunctiva *2cc.* of a 1 in 1,500 cyanide of mercury in the case of a wounded eye, one produced a great outpouring of lymph, and this had proved to be most beneficial, either in preventing infection, or checking it when it had commenced.

D. V. GIRI (Eastbourne) contributed a paper on **milk injections,** and reported on a number of cases he had treated by the method. The result of these injections was, he said, to produce an intensive stimulation of the cells of the body as a whole; there was an increased formation of cells, and greater glandular activity. It was very valuable in hæmorrhagic conditions, and in gonorrhœal ophthalmia in the adult. Contra-indications to its employment were severe traumatism, old degenerative processes, sympathetic ophthalmia, and atrophy of the globe. Iritis, whatever its cause, yielded in a satisfactory way to this treatment.

The Constitution of Future Conventions.

A general meeting of members of the Convention took place on Thursday afternoon to discuss the question whether the next meeting should be of an international character, and be arranged on a pre-war basis. Dr. HOWE proposed, Dr. RING seconded, and the meeting adopted, the following resolution:

"That this Convention of English-speaking ophthalmological societies hereby empowers its President, Mr. E. Treacher Collins, to appoint a standing committee of five, of which he shall be chairman. The duty of this Committee shall be to obtain such coöperation as is possible from representatives appointed by various national ophthalmological societies in promoting matters of international ophthalmological interest, for example: (a) Aiding or initiating measures by international intercourse likely to prevent the dissemination of diseases of the eye and the lessening of hereditary blindness; (b) the establishment of uniform methods of recording results

of visual tests and other matters pertaining thereto; (c) the interchange of ophthalmological literature; (d) the promotion of international ophthalmological congresses."

A further resolution, thanking the English organizers for what they had done, was cordially passed.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M. D., F. A. C. S., New Rochelle, N. Y.

(*Continued from November issue.*)

V.—THE LIDS AND LACRIMAL ORGANS.

104. Beauvieux and Pesme. **La dacryoadenite tuberculeuse, tuberculose attenue de la glande lacrymale.** *Archiv. d'Ophtalm.*, xxxix., 1922.

105. Blaskovicz, v. **Cause of senile entropion.** *Klin. Monatsbl. f. Augenheilkunde*, lxix., p. 136.

106. Blaskovicz, v. **A new operation for senile entropion.** *Ibid.*, lxix., p. 138.

107. Blaskovicz, v. **Operation for ptosis.** *Ibid.*, lxix., p. 682.

108. Braun. **A peculiar form of epicanthus with congenital ptosis.** *Ibid.*, lxviii., p. 110.

109. Carrere. **Local eosinophilia in dacryocystitis.** *Archiv. d'Ophtalmologie*, 1921, p. 233.

110. Duverger. **Techniques operatoires pour l'ablation du sac lacrymal.** *Ibid.*, xxxix., 1922.

111. Van Duyse and van Lint. **Cyste congenital de la glande lacrymal orbitaire.** *Ibid.*, xxxix., 1922.

112. Filatow. **Plastic operations with round pedicles.** *Klin. Monatsbl. f. Augenheilkunde*, lxviii., p. 124.

113. Francis, L. M. **Malignant lymphoma of the lacrimal gland.** *American Journal of Ophthalmology*, March, 1923.

114. Gallenga. **Del "segno di Carlo Reymond" nel corso di talune flogosi del segmento anteriore del globo oculare.** *Periodico d'oculistica*, 1922, No. 5.

115. Greenbaum, S. S. **Isolated favus of the eyelid. Report of a case.** *American Journal of Ophthalmology*, January, 1924.

116. Hessberg. **A congenital familial entropion of both lower lids.** *Klin. Monatsbl. f. Augenheilkunde*, lxviii., p. 120.

117. DE LAPERSONNE and CASTERAN. **Furonculose palpebrale et vaccines.** *Archiv. d'Ophtalmologie*, xxxix., 1922, No. 11.

118. MUELLER. **New operation for entropion.** *Klin. Monatsbl. f. Augenheilkunde*, lxix., p. 288.

119. RAUBITSCHEK. **A new operation for entropion.** *Ibid.*, lxviii., p. 618.

120. RUBBRECHT. **La dacryocysto-rhinostomi.** *Archiv. d'Ophtalmologie*, 1921, p. 165.

121. STREBEL. **Small cystic change of the lower lacrimal gland as the cause of epiphora.** *Archiv f. Augenheilkunde*, xcii., p. 24.

122. WEEKERS and HALKIN. **Hyperemie paroxistique recidivante des paupières.** *Archiv. d'Ophtalmologie*, 1921, p. 641.

123. WHITHAM, L. B. **Teratoma of the lacrimal gland.** *American Journal of Ophthalmology*, September, 1923.

BLASKOVICZ (105, **Cause of senile entropion**) presents the following explanation of the origin of senile entropion. The eyeball is pressed back into the orbit by the pressure of the upper lid, while at the same time the orbital fat about the eyeball is forced forward and outward, and the tarsus is rotated about its longitudinal axis so that the lashes are turned in. All of these displacements are possible only when the tarso-orbital fascia is not sufficiently tense because of too slight an innervation of Mueller's muscle, which is supplied by the sympathetic. The author adduces as an evidence of the correctness of this theory the fact that cocaine hinders the formation of an entropion, because it irritates the sympathetic, while holocain exerts no such preventive influence.

The operation devised by VON BLASKOVICZ (106, **New operation for senile entropion**) is supposed to increase the tension of the tarso-orbital fascia and remove the tarsis from the eyeball. An incision parallel to the margin of the lid is made through the skin about the middle of the tarsus extending toward the temple and curving upwards. At its temporal end a triangular piece of skin with its apex downwards is excised. The skin of the lid is dissected up as far as the infraorbital margin and drawn upwards by the suturing of the temporal triangle. After removal of the tarsal portion of the orbicularis the skin is sutured. He claims that through its involvement in the scar the tarso-orbital fascia is made tense.

MUELLER (118, **New operation for entropion**) excises a triangular piece of conjunctiva and tarsus the apex of which

reaches almost to the intermarginal space. The base line of 4 to 5*mm* is formed at the lower margin of the tarsus. The wound margins are drawn together by a suture at the lower margin of the tarsus. Bandage for one day. The suture usually comes away of itself.

RAUBITSCHEK'S (119, **New operation for entropion**) new operation is a modification of Snellen's modification of Stretfield's operation. Stretfield excised a wedge from the tarsus together with the overlying skin and muscles, and let the wound granulate. Snellen excised a similar wedge from the tarsus and closed the wound with sutures. Raubitschek excises a wedge-shaped piece of the tarsus in like manner and closes the wound with silk threads placed obliquely so as to seem to meander, "die Fadenführung die Form eines Mäanders erhält." Finally the threads are brought out at the commissure of the lids and not tied. After treatment consists of tightening the sutures subsequently if necessary and their removal on the fourth day.

HESSBERG (116, **Congenital familial entropion of both lower lids**) observed a congenital entropion of both lower lids in two sisters. The edges of the lids were turned in, the lashes lying in the conjunctival sac. Below the edge of the lid was a swollen fold of skin. The children showed no other anomalies of development, but were perhaps somewhat tubercular. At the operation no anatomical anomaly was found in the lower lids which could act as the cause of the entropion.

VON BLASKOVICZ (107, **Operation for ptosis**) incises the conjunctiva of the everted lid along the convex margin and introduces three double armed threads in the upper lip of the conjunctival wound, the needles directed toward the tarsus. The conjunctiva is then dissected up to the fornix. The threads are then passed through the levator, in its middle, outer and inner portions, and the loops tied. Then a small piece of the tarsus is excised, the levator is dissected free on its anterior surface and drawn down by means of the three threads fastened to it. The threads in the tarsal conjunctiva are then passed through the levator and through the skin where they are tied over rolls of gauze. The levator is shortened before the fixation.

BRAUN (108, **Peculiar form of epicanthus with congenital**

ptosis) describes a case of ptosis associated with the following condition at the inner canthus. A fold of skin started from the lower lid and arched upward about the end of the upper lid, not covering the canthus, but shoving it toward the temporal side so as to increase its distance from the bridge of the nose. The palpebral fissure was shortened and made oblique.

FILATOW (112, **Plastic operation with round pedicles**) first prepares a round pedicle by dissecting up a strip of skin between two parallel incisions and suturing the edges, leaving the attachment secure at both ends. The gap in the skin is closed by sutures. Three weeks later the plastic operation is performed, the flap to be transplanted being formed at one end of this round pedicle and used to cover the defect. Union is complete in from ten to fifteen days and then the pedicle is cut away. The advantages claimed are better nutrition for the flap and less danger of infection.

GALLENGA (114, **Carlo Reymond's sign in many inflammations of the anterior portion of the eye**) says that in the acute stage of many inflammations of the eye the secretion of the Meibomian glands stops, to change into a hypersecretion as soon as the inflammation of the eye begins to retrogress. This he calls Carlo Reymond's sign. Gallenga's explanation of this phenomenon is that many inflammations of the anterior segment of the eye are associated with a slight swelling of the lids and a certain degree of infiltration of their margins which mechanically obstructs the escape of the secretion from the Meibomian glands. With the decline of the inflammation in the eye the circulatory conditions in the margin of the lid become regulated so that the excretory ducts of the glands are freed and the retained secretion is allowed to escape in large quantities. As this increased secretion is accustomed to accompany the regenerative process in the eyeball, this symptom may be regarded as a prognostically favorable sign.

The lesion in GREENBAUM'S (115, **Isolated favus of the eyelid**) case was confined to the right lower lid near its margin. The clinical appearances were the same as those found in other parts of the body, that is, a slow growth in the form of typical, sulphur yellow, cup-shaped crusts called scutula. The diagnosis was confirmed by mycologic and cultural studies.

ALLING.

DE LAPERSONNE and CASTERAN (117, **Furunculosis of the lids and vaccine**) obtained good results in two bad cases of furunculosis of the lids from the use of a polyvalent vaccine from staphylococci, streptococci and pyocyaneus bacilli. From 1 to 4*ccm* were injected three or four times at intervals of a few days. They describe the way in which the vaccine is made, and consider it valuable in abscess of the orbit and metastatic ophthalmia. It produces a severe general as well as local reaction.

WEEKERS and HALKIN (122, **Recurrent hyperæmia of the lids**) include in one clinical picture several conditions which have received such names as blepharoptosis, blepharochalazia, chalazodermia, dermodolysia, etc. The final result is atrophy of the lid with ptosis. The case reported was that of a boy 15 years old who at the age of 8 began to suffer at night from a spontaneous painful swelling of the left upper lid. In the course of the following day the lid became normal. Such attacks recurred regularly for years without visible cause. Finally the lid became thickened, the attacks less frequent. During the last attack blood appeared behind the upper lid. At the time of examination the eyeball was normal. The lid was in a state of ptosis, its skin lax, in folds, discolored reddish violet by dilated veins, and with enlarged sebaceous glands. The palpebral conjunctiva was very vascular up to the fornix. The lower lid was not affected. Examination of an excised portion showed telangiectases and proliferation of the blood-vessels, dilated lymph spaces, absence of elastic fibers, and a thinned epidermis. The etiology is obscure, but may be neropathic as such patients are very easily excitable.

BEAUVIEUX and PESME (104, **Tuberculous dacryoadenitis**) have collated about thirty cases, together with one observed by themselves. The disease runs a painless course, and the first indication the patient has of its presence is a hard tumor with irregular surface which causes a bulging in the outer portion of the upper lid and increases in size. It has no influence on the position of the eyeball and causes no functional disturbances. The etiology is obscure in the majority of cases. The prognosis is good, as the course is mild and spontaneous recovery occurs. The formation of a cold abscess has been observed once in the rare caseous form, but is not to be feared

in the sclerotic form. The authors incline to believe this to be a primary disease of the gland of hæmatogenous origin. Differentiation from adenoma and other tumors can be made only from the histological characteristics. As an accurate clinical diagnosis is impossible and as the operation is easy and without danger the extirpation of the gland should be recommended in all cases.

VAN DUYSE and VAN LINT (111, **Congenital cyst of the orbital lacrimal gland**) observed a slight exophthalmos with œdema of the upper lid in a new born infant. Within five days the cornea was on a level with the alæ of the nose. Fluctuation in the region of the upper lid and the exophthalmos disappeared after aspiration of a fluid resembling ascites, but ten days later had returned to the same degree. Extirpation of the cyst was followed by perfect recovery. The cyst was unilocular, composed of connective tissue and lined with cylindric epithelium. There were no signs of inflammation to be found in its neighborhood.

FRANCIS (113, **Malignant lymphoma of the lacrimal gland**) removed a tumor which filled the lacrimal fossa and was firmly adherent to the periosteum. The tissue presented the picture of a malignant lymphoma.

WHITHAM (123, **Teratoma of the lacrimal gland**) removed a tumor about 1.5 by 8*cm* in size which proved to be a teratoma. It consisted of yellow fatty tissue embedded in which was a tooth-shaped mass of bone.

STREBEL (121, **Small cystic change of the lower lacrimal gland as the cause of epiphora**) says that the diagnosis of this condition is easy though seldom made, because the ophthalmologist is too easily inclined to think only of inspecting and testing the function of the efferent lacrimal passages in cases of epiphora. Treatment consists of opening the little cysts.

CARRERE (109, **Local eosinophilia in dacryocystitis**) found mononuclear, round eosinophile cells deposited as a concentric zone in the pericystic connective tissue about a lacrimal sac which had been extirpated because of dacryocystitis, at a little distance from the vessels and the usual infiltration of the mucous membrane with leucocytes or lymphocytes. This condition is to be found in various forms of dacryocystitis and at all ages, but is most marked in chronic cases that have

lasted many years. Polynuclear leucocytes are often absent. The eosinophile cells act as phagocytes, aiding the polynuclear neutrophile cells in the various layers of the mucosa.

DUVERGER (110, **Extirpation of the lacrimal sac**) divides the operations for removal of the lacrimal sac into three, each to be performed in accordance with the indications. 1. Simple excision of the sac in chronic dacryocystitis which is without any perivascular complications. 2. Excision of the sac together with its fibrous surroundings after phlegmonous inflammations. 3. Removal together with the fibrous and the bony surroundings, so far as they are diseased, in tuberculous infection. The technique of the first operation is given thus: Skin incision *2mm* from the inner canthus, *1cm* upwards, 2 or *3cm* downwards, then curving parallel to the lower margin of the orbit. Laying bare the superficial fascia by dissection of the inner flap of the wound to *1mm* to the nasal side of its insertion into the bone. This tissue is then cut through not in the skin incision, but rather to its median side. The outer surface of the sac is first laid bare and a retractor inserted, one branch holding the outer lip of the fascia. In laying bare the inner wall hemorrhage usually occurs from the opening of a venous sinus found there. The cupola is separated by dissection from within outwards. After dissecting up the posterior wall the sac is cut off as deeply as possible at the opening of the nasolacrimal canal. The mucous membrane of this canal is then destroyed by means of the thermocautery, and the wound is sutured.

In the second operation the skin incision is more extensive above and below, while the tissues under the inner lip of skin are cut through to the bone. The removal of the lacrimal sac begins this time on its inner surface, working toward the anterior lacrimal crest. The periosteum is taken away together with the sac. After the extirpation the field of operation must be carefully examined to see that no remains have been left behind. Then the wound is sutured, perhaps with drainage.

In the third operation the skin incision starts *3mm* from the inner canthus, runs *2cm* upwards, 5 to *6cm* downwards, embracing any fistula that may be present. The outer lip of the wound is dissected up to the limit of the sound tissue, the inner is made down to the bone. The lacrimal bone and the

anterior ethmoid cells must be carefully investigated, as well as the nasolacrimal canal. These, as well as the cupola of the lacrimal sac and the inner surface of the wound, are cauterized, the outer wall of the wound cavity being left untouched. The skin wound is then carefully sutured to avoid secondary infection. After each of these operations it is recommended to destroy the canaliculi.

Dissatisfied with the results of Toti's operation RUBBRECHT (120, **Dacryocystorhinostomy**) has used the following method in 150 cases with good results. Local anæsthesia in the infraorbital canal and along the inner wall of the orbit to the depth of about *3cm.* Incision *2mm* above the inner canthus about *3mm* to the inner side along the anterior lacrimal crest down to the bone. The periosteum with all the soft parts lifted up with a raspatory. Resection of the upper part of the bony lacrimal canal by means of Cetelli's forceps, laying bare the nasal mucous membrane, which is incised to form flaps. These, after extirpation of the membranous nasolacrimal canal, are united with the lower margin of the lacrimal sac by two fine catgut sutures. Iodoform gauze carefully introduced through the nose holds the flaps in good position for six or seven days, after which the gauze is removed.

VI. THE MOTOR APPARATUS OF THE EYE.

124. BIELSCHOWSKY. **Disturbances of fusion.** *Bericht d. ueber 7 Kongress f. experiment. Psychol.*, Marburg, 1921, p. 105.

125. BIELSCHOWSKY. **Disturbances of fusion and their treatment.** *Archiv f. Augenheilkunde*, xcii., p. 117.

126. BIELSCHOWSKY. **Operative treatment of old paresis of the trochlearis.** *Klin. Monatsbl. f. Augenheilkunde*, November, 1921, p. 611.

127. BORRIES. **Variation in the caloric reaction of nystagmus.** *Monatsbl. f. Ohrenheilk. u. Laryngo-Rhinol.*, lvi., p. 30.

128. CALKINS, J. W. **A muscle "folder" for strabismus.** *American Journal of Ophthalmology*, November, 1923.

129. CANTONNET. **Double paresis of the abducens in tabes.** *Archiv. d'Ophtalmologie*, 1922, xxxix., No. 6.

130. DELORD. **Amblyopia in strabismus.** *Ibid.*, 1921, p. 597.

131. DELORD. **Persistent ocular paralyses in the course of epidemic encephalitis.** *Ibid.*, 1922, xxxix., No. 9.

132. FISCHER. **Movements of the eyeball excitable through the ear.** *Monatschr. f. Ohrenheilk. u. Laryng.*, lv., p. 764.

133. FISCHER, B. **The peripheral and central vestibular apparatus in multiple sclerosis.** *Zeitschr. f. d. ges. Neurologie u. Psychiatr.*, lxxvi., p. 42.

134. Fuss. **Absolute and relative convergence and divergence movements.** *Archiv f. Ophthalmologie*, cix., p. 428.

135. Knick, A. **Paralysis of the abducens in otitis media.** *Zeitschr. f. Hals-, Nasen- u. Ohrenheilk.*, iii., p. 136.

136. Kremer. **Operative treatment of old pareses of the trochlearis.** *Klin. Monatsbl. f. Augenheilkunde*, November, 1921, p. 600.

137. Lagrange and Pesme. **Paralysie de l'oculomoteur externe du côte droit et anisocorie par myosis du même côte après rachianesthésie.** *Archiv. d'Ophtalmologie*, 1922, xxxix., No. 9.

138. Landolt, E. **A study of strabismus.** *American Journal of Ophthalmology*, February, 1923.

139. Li, T. M. **Congenital total bilateral ophthalmoplegia.** *Ibid.*, October, 1923.

140. Ohnacker. **Nystagmus synchronous with the pulse.** *Zeitschr. f. Hals-, Nasen- u. Ohrenheilk.*, ii., p. 401.

141. Quist. **Recurrent paralysis of the abducens.** *Zeitschrift f. Augenheilkunde*, xlvii., p. 289.

142. Rateau. **Traumatic paralysis of the superior oblique by contrecoup.** *Archiv. d'Ophtalmologie*, 1922, xxxix., No. 12.

143. Terrien. **Traumatic ophthalmoplegias.** *Ibid.*, 1922, xxxix., No. 2.

144. Young, G. **Subconjunctival advancement.** *British Journal of Ophthalmology*, 1922, No. 7.

According to Bielschowsky (124, **Disturbances of fusion**) fusion movements are not true reflexes, although they are not under the control of the will, because stimuli induce a motor effect only under the influence of the attention. Disturbances of fusion may appear more or less suddenly as the result of great psychical exhaustion or a psychical shock while the sensory apparatus remains intact. Transition stages exist from the normal to the pathological condition. In a second group of disturbances the entire fusion apparatus fails; the two eyes obey the impulse to harmonious movement, but the unison of the two eyes is sensorily absent. Absolute absence of fusion is irreparable and depends on an organic defect.

The same writer says (125, **Disturbances of fusion and their treatment**) that there are three types of these disturbances. To the first group belong the cases in which diplopia appears, usually suddenly, which is not of paretic origin but is dependent on the cessation of the motor reaction to disparate stimulations of the retina. This disturbance is usually obstinate. The second group is met with more frequently. The subjective characteristic is muscular asthenopia. The power of fusion is unimpaired, but its action induces subjective troubles.

The third group is the smallest, illustrated by Bielschowsky by a single case of spasm of the accommodation in which the associated convergence innervation was used up in concealing an exophoria. So far as treatment is concerned he recommends prisms when the heterophoria is of low degree, but diplopia exists in consequence of failure of the fusion mechanism. But they should be given only after general treatment and fusion exercises have proved unavailing, because the prisms conserve a weakness of fusion. When the heterophoria is 6° or more operative procedures should be considered.

KREMER and BIELSCHOWSKY (136 and 126, **Operative treatment of old pareses of the trochlearis**) report concerning fourteen cases of trochlear paralysis treated by Bielschowsky's operation of setting back the inferior rectus of the other eye. The results are noteworthy. As a rule diplopia is obviated in the important lower half of the visual field. An overeffect, which was formerly always feared, has not yet been seen. It is of interest that among the cases were several with torticollis, the real cause of which had not been recognized, who had long worn orthopædic appliances without benefit.

RATEAU (142, **Traumatic paralysis of the superior oblique by contrecoup**) reports a case of blow on the forehead that caused an isolated paralysis of the superior oblique, which disappeared at the end of fifteen days. Aside from tenderness in the region of the trochlea nothing abnormal was found.

CANTONNET (129, **Double paresis of the abducens in tabes**) reports eight observations. In six of these the condition has existed four years without change. Therefore pareses of the ocular muscles may be not transient, as has been claimed. Cantonnet thinks the superficial position of the abducens nucleus is answerable for the bilateral paresis, which has been observed also in diabetes and in lumbar anæsthesia.

The origin of the paresis of the abducens which so often accompanies an otitis media has repeatedly been a subject for discussion. It has been thought a reflex paralysis, has been ascribed to a toxic neuritis, to ostitis of the apex of the petrous portion of the temporal bone, and most frequently to a circumscribed meningitis. KNICK (135, **Paralysis of the abducens in otitis media**) has examined the cerebro-spinal fluid in five cases. In one case, in which there were clinical symptoms of

meningitis, there was an increase of cells and albumin in the fluid, but in the other cases the fluid was normal. The pareses disappeared in from three to twelve weeks.

LAGRANGE and PESME (137, **Paralysis of the right abducens with anisocoria due to miosis of the right eye after lumbar anæsthesia**) observed these symptoms in a woman 39 years old in whom lumbar anæsthesia had been produced for surgical purposes. Other accompanying symptoms were pain in head and neck and severe vomiting. The writers are inclined to ascribe the paralysis of the abducens to an injury of the nucleus by the anæsthetic introduced into the lumbar sac, and the miosis to syphilis, from which the patient was suffering.

QUIST (141, **Recurrent paralysis of the abducens**) reports a case of this nature met with in a student 20 years old, which ran a course analogous to that of the corresponding affection of the oculomotorius. For four years the patient had suffered at intervals of four or five months from attacks of headache which lasted from twelve to twenty-four hours with diplopia which lasted eight or ten days and then disappeared. A thorough general examination gave a completely negative result.

DELORD (131, **Persistent ocular paralyses in the course of epidemic encephalitis**) reports three cases. In the first there was paresis of the left internal rectus, the left trigeminus, and the right facial, with subsequent anisocoria. Another had micropsia, paralysis of the hypoglossus, and convergence with diplopia. The third showed total paralysis of both internal recti with no other symptoms.

LI (139, **Congenital total bilateral ophthalmoplegia**) reports the cases of three children out of a family of five who showed practically complete bilateral paralysis of all the eye muscles. He regards the lesion as nuclear and probably hereditary. He thinks that operations would not be advisable.

ALLING.

TERRIEN (143, **Traumatic ophthalmoplegias**) reports two cases. The first was one of the rare cases of an isolated lesion in the region of the superior orbital fissure. The temporary paralysis of all the motor and sensory nerves passing through this fissure together with slight exophthalmos makes the theory of a hæmatoma at the apex of the funnel of the orbit more prob-

able than that of a splintering fracture at the fissure alone. The visual field of the eye was normal, but the vision was reduced by little hemorrhages in the macula. Simultaneously a neuroparalytic keratitis developed, such as is usually observed in affections of the Gasserian ganglion. The second case was associated with a fracture of the base of the skull, which presented characteristic features of a fracture through the temporal bone with hemorrhages at the base.

FUSS (134, **Absolute and relative convergence and divergence movements**) distinguishes between an absolute and a relative innervation of convergence and divergence. The nullpoint of the innervation he places in the so-called relative postion of rest, *i.e.*, the position in which the position of the eyes is fixed wholly by the anatomical structure and the tone of the muscles. That the parallel position of the eyes for distance is not the position of rest, but is taken under the influence of a convergence-divergence innervation is Fuss's contention. The theoretical outpouring of the writer culminates in this, that the fusion movements seem to render it logically necessary that there should be a divergence center as well as a convergence center.

LANDOLT (138, **A study of strabismus**) emphasizes the fact that concomitant strabismus is not alone a fault in one muscle but a binocular affection. He does not believe in amblyopia ex anopsia but thinks that strabismus is due to the congenital amblyopia. All will not concur in his statements that after tenotomy a divergence strabismus nearly always follows and that the caruncle is retracted with protrusion of the eyeball, nor is he quite correct in saying that the motility of the squinting eye in convergence strabismus is not increased inwards. In all cases even the paralytic he advocates advancement and resection of the weak muscle and never does a tenotomy.

ALLING.

DELORD (130, **Amblyopia in strabismus**) sought to improve the vision and function of the amblyopic eye in strabismic children ranging from 4 to 9 years of age by occlusion of the better one and correction with glasses when necessary. In most cases the vision improved and, when the improvement continued up to the tenth year, appeared to be permanent. The squinting position of the amblyopic eye was often bettered.

CALKINS (128, **A muscle "folder" for strabismus**) uses a stiff wire bent in shape like a hairpin with the free ends fitting into a handle. The hairpin loop is put in place so that the exposed muscle lies between its two prongs. By revolving the instrument the muscle is rolled up about the loop for a number of times sufficient to produce the desired amount of shortening. The rolled up muscle is then sutured after which the loop is removed.

ALLING.

YOUNG (144, **Subconjunctival advancement**) makes a small opening in the conjunctiva and separates the muscle completely from the sclera with a small knife. At two opposite points the conjunctiva and belly of the muscle are pierced by two strong silk threads in doubled loops and fastened to corresponding places in the vicinity of the limbus. In this way the anterior surface of the muscle is folded upon itself and grows together, while its posterior surface becomes reattached to the sclera. Both eyes are bandaged for five days, the sutures allowed to remain in for two weeks. A disadvantage is that the folding of the muscle creates a large prominence, but after a few months this is no longer to be seen. The effect produced is very great and is dependent on the distance of the point of puncture from the cornea and the tension exerted in the tying of the sutures.

BORRIES (127, **Variation in the caloric reaction of nystagmus**) has observed in six cases that when the head was bent 90° backward or forward the inversion of the caloric nystagmus almost always induced did not take place. Two of the patients had serous labyrinthitis, two an injury of the head, one vestibular neuritis and one epidemic meningitis. The galvanic and rotation nystagmus may be normal or not.

FISCHER (132, **Movements of the eyeball excitable through the ear**) reports some unusual cases in which by pressing upon the tragus he induced a strong spasm of the convergence and accommodation. In others this phenomenon was induced by the production of a rotation nystagmus and persisted as long as the nystagmus lasted. The spasm was accompanied by giddiness and a tendency to fall backwards. Fischer considers this evidence that in certain predisposed individuals the labyrinth should be considered a hysterogenous zone.

FISCHER (133, **The peripheral and central vestibular apparatus in multiple sclerosis**) found spontaneous nystagmus in 75% of his cases, in 60% only when the eyes were turned to one side, but believes that this percentage may be too high. A change in the form of nystagmus was observed repeatedly. The caloric test gave a normal result in 58.5%, a stronger nystagmus than normal in 31.5%. In four cases the caloric reaction failed. Once he saw a slow deviation in place of the vestibular nystagmus.

OHNACKER (140, **Nystagmus synchronous with the pulse**) reports four cases of this nature with fistula of the labyrinth in otitis media, one with formation of granulations after a radical operation. In three cases compression of the carotid induced a nystagmus toward the opposite side which frequently became reversed after removal of the pressure. Ohnacker thinks that a movement of the endolymph is not necessary to the production of this nystagmus. The vestibular symptoms are not present in all cases of fistula of the labyrinth and are plainly favored by disturbances in the circulatory apparatus.

VII.—INTRAOCULAR FLUIDS AND GLAUCOMA.

145. ALTLAND. **Report of a case of congenital hydrophthalmos treated by trephining.** *Zeitschrift f. Augenheilkunde*, October, 1922, p. 55.

146. AMSLER. **Reaction générale et reaction locale oculaire.** *Rev. génér. d'Ophtalmologie*, 1921, No. 11, p. 477.

147. ASCHER. **Tonometry with varying air pressures.** *Naturforscher Versamml.*, Leipsic, 1922.

148. BARTELS. **Chronic glaucoma hereditary at the climacteric period.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 298.

149. ELLIOT, R. H. **The mists and halos of glaucoma.** *American Journal of Ophthalmology*, January, 1923.

150. ERÖDS. **Cyclodialysis or trephining.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 175.

151. FILBRY. **The tension and pharmacodynamic behavior of the eye after injection of alcohol into the Gasserian ganglion.** *Ibid.*, October, 1922.

152. GOLDENBURG, M. **Iridotasis operation for glaucoma.** *American Journal of Ophthalmology*, May, 1923.

153. HEYMANS-MAY. **Rapports entre la tension intra-oculaire et le développement des tumeurs du tractus uveal.** *Archiv. d'Ophtalmologie*, 1921, No. 8, p. 479.

154. LEVINSOHN. **Pathogenesis of glaucoma.** *Deutsche med. Wochenschr.*, 1922, p. 340.

155. Lo Cascio. **Sui fermenti dei tessuti e dei liquidi oculari.** *Annali di ottalmologia e clinica oculistica*, 1922, Nos. 3–5.

156. Pick. **Malignant glaucoma.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 290.

157. Samoyloff. **Changes of glaucomatous scotomata in fluctuations of the intraocular tension.** *Klin. Monatsbl. f. Augenheilkunde*, lxix., p. 59.

158. Terrien. **L'eclatement spontane du globe oculaire.** *Archiv. d'-Ophtalmologie*, 1921, No. 4, p. 205.

159. Teulieres and Pesme. **De la methode fistulisante dans le traitement du glaucoma chronique.** *Ibid.*, 1921, No. 4, p. 215.

160. Ulrich. **Indications for glaucoma operations.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 289.

161. Weekers. **Le drainage permanent du vitre dans le glaucome.** *Archiv. d'Ophtalmologie*, 1922, xxxix., No. 5.

162. Weekers. **Elimination des liquides intra-oculaires chez l'homme.** *Ibid.*, xxxix., No. 9, 1922.

163. Weisenberg. **Experiences with cyclodialysis.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 290.

164. Wilder, W. H. **Some observations on iridotasis in the treatment of glaucoma.** *Journal A. M. A.*, December 22, 1923.

165. Yoshida, Y. **Acute glaucoma following Heine's cyclodialysis.** *American Journal of Ophthalmology*, May, 1923.

In the course of three months Amsler (146, **General and local ocular reaction**) studied the tension of an eye suffering from detachment of the retina and iridocyclitis using Schiötz's tonometer, and found that it fluctuated between − 3 and + 8. After injections of salt solution the tension sometimes rose to + 15 or 17Hg. The patient had an attack of acute bronchitis with a temperature up to 40°, and a few hours later the tension of this eye had risen to ½ 45*mm* Hg, although that of the other, likewise myopic, eye remained normal. The temperature and tonometer curves fell synchronously. A similar condition was observed after a diagnostic injection of tuberculin in a young man 17 years old with tubercular uveitis. No influence of the body temperature upon the eye tension could be found in febrile patients without eye diseases.

Ascher (147, **Tonometry with varying air pressure**) studied the relation between air pressure and intraocular pressure on both man and animals in the pneumatic cabinet. Schiötz's tonometer and the open manometer tube showed no material changes in the intraocular pressure, but when the anterior chamber of a rabbit was united with a manometer closed at the end there was a diminution of the intraocular pressure

when the air pressure was increased, and an increase of the intraocular pressure when the air pressure was decreased. The possibility of an onset of glaucoma at great heights is therefore not absolutely excluded.

FILBRY (151, **Tension and pharmacodynamic behavior of the eye after injection of alcohol into the Gasserian ganglion**) observed total anæsthesia and reduction of tension 4*mm* of the eye on the side on which the injection was made, as compared with the other eye. Its pupil was also dilated more widely by cocaine—adrenalin and by atropine than that of the other.

LO CASCIO (155, **The ferments of the tissues and the fluids of the eye**) shows that both the tissues and the fluids of the eye possess the ability to oxidize specific reagents. Pyrogallol is oxidized more quickly and strongly by the vitreous and the aqueous than by the other tissues. The reverse of this is true with regard to paraphenylendiamin. It appears that the power of the aqueous and vitreous to oxidize is of a thermostabile nature, while that of the other tissues is affected to a certain degree by the temperature. The aqueous, vitreous and to a less degree the lens are able to break up hydrogen peroxide with the production of oxygen; the strength of this catalytic power is to that of the blood serum as 1:9. With regard to the reducing ferments, both the tissues and the fluids are able to reduce methyl blue to leocomethyl blue by warming or by shaking in the air; this power of reduction is of a thermostabile nature and is to be ascribed to the presence of special enzymes. The retina of an animal which has been in the dark for a long time has a greater power of reduction than that of an animal which has long been exposed to light. With regard to ferments that break down albumin, the aqueous and vitreous have little proteolytic power upon the albumin of the lens, yet antitryptic ferments are present neither in the lens nor in the fluids of the eye, as they are in the blood serum. The resistance of the lens to the proteolytic ferments therefore depends wholly upon its capsule.

WEEKERS (162, **Elimination of the intraocular fluids in man**) injected a minute quantity of India ink into a healthy eye which was destined for enucleation because of a malignant tumor in the orbit. No change took place in the tension. The distribution of the particles in the vitreous could be observed

with the ophthalmoscope, but none could be seen in the anterior chamber up to the time of enucleation an hour and a half later. Study of the sections of the eye showed a uniform distribution of the ink in the vitreous with a slight increase in front of the hyaloid. In the anterior chamber minute particles were found in the iridocorneal angle, on the posterior surface of the cornea and the anterior surface of the iris. The pectinate ligament was distinctly blackened, especially on its inner side, the veins were invested by a black mantle, but the lumen of the veins and of the canal was perfectly free. The infiltration of the interstitial tissue between the ciliary body and the sclera was very plain. Black deposits were to be found throughout the posterior layer of the iris separated from the pigment layer by the fibers of the dilatator. Only the peripupillary region and the root of the iris were affected on the part of the anterior layers, part of the stomata were rendered visible by the deposited ink. Numerous accumulations of ink were found in the deep layers of the ciliary body next to the epithelium. The marked impregnation of this organ and of the large veins lying in its posterior part indicate that a considerable absorptive process may have taken place here. No deposits were found in the suprachoroideal space, the choroidal and retinal vessels, or the papilla and its neighborhood. It would appear from this experiment that the anterior segment of the eye alone takes part in absorption in confirmation of the classical teaching of the elimination of the aqueous. But it seems also to show elimination in the corneo-scleral tissue, in the ciliary body, and in the vascular system of the iris, which perhaps play secondary but important parts.

HEYMANS–MAY (153, **Relations between the intraocular tension and the development of tumors of the uveal tract**) studied twelve cases, one of sarcoma of the iris, five of the ciliary body, five of the choroid, and one of cholesteatoma of the retina. The tension was subnormal in four, normal in two and above normal in six. He points out that the angle of the anterior chamber may appear to be closed without increased tension being present and is of the opinion that there is no direct relation between the condition of the angle and the position or size of a tumor.

TERRIEN (158, **Spontaneous rupture of the eyeball**) reports

the case of a man 70 years old whose one eye suffered from chronic glaucoma with subluxation of the lens. One night he was suddenly seized with extreme glaucomatous pain which was followed by a spontaneous rupture and hemorrhage. The place of rupture was at the limbus in its outer, lower quadrant; the rupture was 1*cm* long and through it the uveal tissue was prolapsed. The eyeball was enucleated. There was a total detachment of the choroid caused by a hemorrhage from one of the posterior ciliary arteries. The retina was atrophic, the papilla not noticeably excavated. The lens could not be found.

LEVINSOHN (154, **Pathogenesis of glaucoma**) differentiates four groups of causes for glaucoma, the pathogenetic, the predisposing, the exciting and the basal. He considers a true pathogenetic cause to be the infiltration of the anterior outflow passages with broken up constituents of the posterior pigment epithelia, or of the stromal pigment. A predisposing cause is an enlargement of the ciliary body through its mechanical and trophic action. Exciting causes are psychic affections and sensory irritations, which act through the dilatation of the pupil. Favoring conditions are circulatory disturbances, less of the blood pressure than of the quality of the blood and of its distribution.

BARTELS (148, **Chronic glaucoma hereditary at the climacteric period**) reports the case of a woman 45 years of age suffering from simple glaucoma. Her sister, mother and three aunts had the same disease and all were attacked during the climacteric.

ELLIOT (149, **The mists and halos of glaucoma**) points out the importance of these symptoms as helps in the diagnosis of glaucoma and in determining the frequency of attacks of tension of which they are indications. The cause of halos is probably an edema of the cornea in the lamellæ lying immediately underneath Bowman's membrane and not stretching of the cornea as has been suggested. He explains the mists of which patients complain in the morning as due to the fact that the pump-like action of the iris and ciliary body ceases during sleep and hence there is a tendency to rise in tension. Those cases in which the halos are seen later in the day are due to vasomotor disturbances incident to fatigue and exhaustion

of the nervous system. Halos may be artificially produced by looking through glass which has been breathed upon or sprinkled with lycopodium powder. There are a number of phenomena which may be mistaken for the halos of glaucoma, for instance, beams which are seen by the normal eye about the lights at night but they are of spectral nature and are not colored rings. Halos appear also after application of caustic such as nitrate of silver and also when a catarrhal discharge is present in the conjunctival sac. Diffraction phenomenon in the lens may be the cause of this symptom and he records numerous experiments which have been made along these lines. He has found that the diameter of the outer orange red ring of the glaucoma halo varies from 60° 50′ to 11° 54′ and gives the formula to be used in making these measurements.

ALLING.

SAMOYLOFF (157, **Changes of glaucomatous scotomata in fluctuations of the intraocular tension**) finds that if, in glaucoma, the blind spot is drawn out vertically and curves about the point of fixation the ends of the scotoma may encounter peripheral contractions. A shortening of the scotoma appears an hour after the instillation of pilocarpine, and is greatest after two or three hours. The previous size is regained after ten to twelve hours. The enlargement of the scotoma can be held back by continuous use of pilocarpine, to start again as soon as the remedy is stopped. The differentiation of such scotomas as can be made to retrogress in part from permanent injuries is important. The best results are to be obtained in scotomas that start from the blind spot, but no regular result can be obtained from pilocarpine in those connected with the periphery. The size of physiological scotomata, or of those due to other causes than glaucoma, is not influenced by pilocarpine.

ALTLAND (145, **Congenital hydrophthalmos treated by trephining**) reports the case of a 6 years old child whose left eye was blind while the right had a tolerable degree of vision. The tension of the right eye was 45*mm*, of the left 55*mm*. Both were trephined with a large iridectomy. Three months later the tension of each eye was 24*mm*. A year later the child had useful vision.

ERDÖS (150, **Cyclodialysis or trephining**) compares his results in 493 cases of trephining and 53 of cyclodialysis. In

inflammatory glaucoma trephining reduced the tension in 83.8%, cyclodialysis in 44.5%, in simple glaucoma trephining in 85%, cyclodialysis in 33.3%. The proportion in absolute glaucoma was similar. Loss after trephining 0.6%, after cyclodialysis 11.2%. Cyclodialysis he thinks therefore should be restricted to those cases in which trephining has failed, and that trephining should be performed only on eyes in which iridectomy would be expected to fail.

GOLDENBURG (152, **Iridotasis operation for glaucoma**) after five years experience recommends this operation as simple and effective. After dissecting down a flap he makes a small incision (about 4*mm*) well back in the sclera and seizing the iris at its middle tucks it in at both ends of the cut.

ALLING.

The right eye of PICK'S (156, **Malignant glaucoma**) 54 years old patient had been blinded by glaucoma, while the commencing disease in the left eye manifested itself by frequent attacks of color vision, although the vision was perfect and the fields normal. Iridectomy of the right eye did not restore the anterior chamber. Cyclodialysis on the left eye was followed the next day by an attack of glaucoma which did not yield to miotics used for several weeks, but finally improved after an anterior sclerotomy. Cyclodialysis does not appear to be without danger in all cases.

TEULIERES and PESME (159, **Fistulization in the treatment of chronic glaucoma**) prefer Lagrange's sclerectomy, often combined with peripheral iridectomy, to trephining, because they ascribe to the latter such ill results as traumatic cataract, malignant glaucoma, hemorrhage into the vitreous, and late infection.

ULRICH (160, **Indications for glaucoma operations**) raises the question whether trephining is the procedure most without danger in chronic glaucoma with marked contraction of the visual field, and whether any increase of tension is an unquestionable indication for an operation. In three cases with marked contraction of the visual field and large paracentral scotomata trephining caused loss of the central vision, while a case with circular scotoma was benefited by iridectomy. Trephining he therefore considers not without danger. Like iridectomy it may be of benefit, but it may not. Increase of

tension alone is not sufficient to indicate an operation for high tension may persist for years without causing harm. An operation is indicated only when harm has resulted.

WEISENBERG (163, **Experiences with cyclodialysis**) performed this operation twenty-five times on twenty eyes in seventeen patients. The anæsthetic was novocain injected subconjunctivally. There were no special incidents. Seventeen eyes have been under observation longer than six months. Of these seven showed good results, four a reduction of tension but not to normal, and six failures, of which two were benefited later by trephining. The intervention usually does good, particularly in simple and chronic glaucoma. No permanent harm was observed but the reduction of tension was not always quick and lasting. Cyclodialysis is not suited to acute glaucoma, or to people who cannot be under skilled observation. For the latter trephining is preferable. When indicated in chronic glaucoma it has the advantage that it may readily be repeated.

YOSHIDA'S (165, **Acute glaucoma following cyclodialysis**) experience with this operation has revealed a danger which has hitherto received little comment although slight rise of tension has been noted. The only explanation for the acute increase in tension is that the traumatism of the operation produces swelling of the ciliary body.

ALLING.

WEEKERS (161, **Permanent drainage of the vitreous**) has performed the following operation in a few cases of painful absolute glaucoma as a substitute for enucleation. Beneath a conjunctival flap he trephines the sclera in the region of the ora serrata making an opening *2mm* in diameter. Into this opening he introduces a little gold ring with grooved margins. Then he removes whatever has prolapsed of the uvea and closes the conjunctival wound. Soon after the operation a conjunctival cushion appears, the tension becomes normal, the anterior chamber deeper. Eyes thus treated may be preserved without renewed increase of tension and pain.

In forty-eight operations performed by WILDER (164, **Some observations on iridotosis in the treatment of glaucoma**) the tension was reduced for a varying time to ninety per cent. but he does not feel justified in drawing conclusions as to its per-

manency. Whether the drainage is accomplished by drawing away the root of the iris from the filtration angle or by the fact that perhaps the iris acts as a living seton, is undecided. He believes the operation easy to perform and accompanied by a minimum of danger. On the whole he prefers iridectomy in congested glaucoma but thinks that iridotasis gives as good results as any operation in non-congested cases and it is especially indicated in patients with atheromatous vessels where a tendency to hemorrhage is present. ALLING.

(*To be continued*)